VOLUME 7 NUMBER 5

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

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MAY, 1914

PUBLISHERS

AMERICAN MEDICAL ASSOCIATION

CHICAGO, ILL.

Entered as Second-Class Matter, January 4, 1911, at the Post-Office at Chicago, Illinois, Under Act of Congress of March 3, 1879

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American Journal of Diseases of Children

VOL. 7 MAY, 1914 No. 5

THE INTESTINE, IN HEALTH AND CATARRH, AS A PATHWAY OF INFECTION TO THE TUBERCLE BACILLUS *

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To what extent does the intestine afford ingress to the tubercle bacillus is a problem that has received much attention within recent years, and yet, despite the enormous amount of work which has been done, we seem no nearer a definite conclusion. From a survey of the abundant literature on the experimental aspect of this subject there emerge three main questions that demand solution:

1. Is the healthy intestinal mucosa pervious to the organism?

2. Do the bacilli leave behind them absolutely no trace of their passage through the gut?

3. Can primary pulmonary tuberculosis arise from bacilli which have gained admission to the body in this way?

To each of these queries contradictory answers have been given. Calmette,[1] as is well known, holds that all tuberculosis, whether primarily pulmonary or not, arises from bacilli which have entered by the intestinal route. He, in common with Vansteenberghe,[2] Beitzke,[3] Engel and Schlossmann,[4] Nebelthau,[5] Uffenheimer,[6] Bartel[7] and others, believes that the bacilli can easily pass through the healthy mucosa without causing any lesion at the point of entrance, and that even within a few hours after feeding an animal on tuberculous material, the bacilli can be demonstrated in the mesenteric glands, lungs and other distant organs. Orth and Rabinowitsch,[8] who also share this

* From the Physiological Department, Glasgow University. In aid of this research a grant was obtained from the Carnegie Trust.

1. Calmette: Ann. de l'Inst. Pasteur, 1905, p. 612.
2. Vansteenberghe: Ann. de l'Inst. Pasteur, 1910, p. 316.
3. Beitzke: Virchows Arch. f. path. Anat., 1914, p. 225.
4. Schlossmann and Engel: Deutsch. med. Wchnschr., 1906, p. 1070.
5. Nebelthau: München. med. Wchnschr., 1903, p. 1246.
6. Uffenheimer: Arch. f. Hyg., 1906, lv, p. 1; Berl. klin. Wchnschr., 1906, p. 421.
7. Bartel: Wien. klin. Wchnschr., 1905, No. 7.
8. Orth and Rabinowitsch: Virchows Arch. f. path. Anat., 1908, cxciv, 305.

opinion, and who, in order to avoid insufflation, introduced bacilli per rectum, report that they have recovered them from the blood a few hours later.

Cobbett,[9] on the other hand, while admitting that the healthy mucosa is permeable by the tubercle bacillus, and that as a rule the passage of the organism through the wall of the gut causes no local change, denies that the lungs are ever infected in this way except as part of a generalization of the mischief. Cobbett in his work animadverts on the great tendency to insufflation in all feeding experiments, and on account of this fact questions some of the conclusions of the British Royal Commission.

Baumgarten,[10] who was probably the first to attack this problem experimentally, in a paper which he read before the German Pathological Society at Berlin in 1904, states that, although animals can be infected by feeding with tuberculous material, such does not invariably occur; and he most emphatically declares that infection of any organ, even of the mesenteric glands, never occurs without a local intestinal lesion being present. Heller,[10] in the discussion which followed, concurred in the opinion that primary intestinal lesions are usually present, but said that they not infrequently heal, and consequently all trace of the portal of entry may be lost.

Others again, e. g., Most,[11] Klimenko,[12] de Vecchi,[13] and Kuss and Lobstein[14] do not believe, as a result of their researches, that animals can be infected by feeding with tuberculous material, and all these authors lay great stress on the probability that, whenever infection appears to have arisen in this way, it is in reality due to insufflation.

It is very doubtful, however, if many of these experiments justify the authors' conclusions. In perhaps the majority demonstrating the passage of the bacilli through the intestinal wall within a few hours after the ingestion of the infected material, the presence of living bacilli was verified by inoculating guinea-pigs intraperitoneally with emulsions of the organs supposed to contain the bacilli. But even if it be admitted that occasionally a few bacilli pass through the intact intestinal mucosa, and lodge in the mesenteric glands or in some distant organ, it is very questionable if disease would have developed in these organs themselves had the animals been allowed to live. Neisser[15]

9. Cobbett: Jour. Path. and Bact., 1910, xiv, No. 4, p. 563.
10. Baumgarten: Verhandl. d. d. Gesellsch., 1904, ix, 5. He stated in 1904 that he had been carrying out experiments on this subject for twenty years.
11. Most: Berl. klin. Wchnschr. Bd., 1908, xlv, No. 8.
12. Klimenko: Ztschr. f. Hyg., xlviii, 67.
13. de Vecchi: Abstract in Centralbl. f. Bact., 1909, p. 786.
14. Kuss and Lobstein: Tuberculosis, vi, 325.
15. Neisser: Beitr. z. Pathol. u. Therap. d. Syphilis, J. Springer, Berlin.

in his work on experimental syphilis has shown that not only the probability of infection, but also the course of the disease, depend in no small measure on the site of inoculation; hence, although there were sufficient bacilli in these organs to cause tuberculosis when introduced into the peritoneal cavity, there is no justification for concluding that disease of the lungs or mesenteric glands would necessarily have resulted.

Chaussé,[16] who admits that in the dog tubercle bacilli may pass through the uninjured intestinal mucosa, states that those absorbed are held up in the mesenteric glands and do not give rise to lesions under normal conditions. Many of the experiments of these same authors substantiate this view, since it is only a certain proportion of the animals fed on bacilli that develop tuberculosis when watched for prolonged periods.

In not a few of the digestion experiments, which have been carried out to demonstrate the passage of tubercle bacilli through the intestinal wall, the infective material has been introduced by means of the stomach-tube, or it has been mixed with the food and the animal allowed to partake of it naturally. Both methods, however, readily permit of insufflation, and in order to avoid this contingency many procedures, operative and otherwise, have been invented, but these unfortunately only too often have admitted of other fallacies.

Nebelthau introduced the bacilli, enclosed in gelatin capsules, directly into an isolated loop of intestine, but it is evident from his protocols that the intestine could not be described as healthy, since shortly after the commencement of the experiment the bowel became distended, its surface glazed, and the mesentery injected. Vansteenberghe, in his latest investigations, to avoid injury of the stomach from the introduction of the tube, and also to render insufflation less likely, allowed his animals a special food with which the bacilli were mixed. This diet consisted of mashed fresh carrots and moistened bread, but here again it is impossible to say that the experiments were carried out under normal conditions, since he remarks in his communication that in consequence of this special régime many of the animals died.

In the researches of Schlossmann and Engel the bacilli were injected directly into the stomach after laparotomy, but as Fraser,[17] Ravenal and Reichel[18] have shown, and as I[19] have also demonstrated, it is impossible to perform this operation (and the same applies to gastropexy) without infecting the peritoneum; hence conclusions founded on any such measures are valueless.

16. Chaussé: Acad. d. Sc.: Abstr. Ztschr. f. Kinderh., 1911, i, 687.
17. Fraser: Jour. Exper. Med., 1913, xvii, 362.
18. Ravenal and Reichel: Jour. Med. Research, 1908, p. 1.
19. Findlay: Ztschr. f. Kinderh., May, 1913.

Orth and Rabinowitsch, as mentioned above, introduced the bacilli per rectum, but in view of the recent work of E. Burville Holms[20] on "The Finding of Alleged Tubercle Bacilli in the Circulating Blood," which draws attention to the fact that many of the acid-fast organisms hitherto discovered in the blood were obtained from the distilled water used in lysing the corpuscles, and not from the blood itself, their results require confirmation.

I do not suppose anyone will deny that organisms are probably always gaining entrance to the economy, whether or not through some *locus minoris resistentiae* in the form of a slight abrasion of the skin or mucous membrane of a viscus it is impossible to say, but in all likelihood they enter in such small numbers that so long as the resistance of the tissues is at a normal level no harm takes place. Were it otherwise one would imagine that the *Bacillus coli,* a beneficent microbe while within the lumen of the bowel, but a most dangerous parasite when beyond those confines, would much more frequently migrate into the tissues and cause inflammatory and suppurative troubles. Personally I am inclined to the view that some injury or weakness of this, the body's first line of defense, is essential for the admission of organisms in sufficient numbers to cause disease.

The mucous membrane of the intestine is a highly specialized tissue, and will assuredly exert a selective action. The work of Cobbett,[9] of Montgomery,[21] and also that by myself[22] and others on "Experimental Pulmonary Anthracosis," support this contention, since it has been shown that a foreign substance, such as soot, is not absorbed, while iron, a normal constituent of the tissues and thus more of the nature of a food and of use to the economy, is. Between such an inert substance as soot and the tubercle bacillus there is, it must be admitted, one very radical difference. The tubercle bacillus is a living organism and, should only one germ gain admission, it possesses, in virtue of its vitality, the ability to multiply, and thus has a potentiality for the production of disease. This would in all probability occur much more frequently were it not for the second line of defense in the tissues, where the microbes, if not too numerous or too virulent, are overcome. In the case of the skin one is dealing with a highly protective membrane, and though experiments have been conducted to prove the passage of tubercle bacilli through this tissue while in a healthy condition, it is doubtful if such was really the case. Koenigsfeld[23] rubbed emulsions

20. Holms, E. Burville: Sixth Ann. Rep., Henry Phipps Inst., Philadelphia, 1912, p. 127.
21. Montgomery: Jour. Med. Research, 1910, xxiii, No. 1.
22. Findlay: Ztschr. f. Kinderh., 1911, ii, 293.
23. Koenigsfeld, Centralbl. f. Bact., 1911, lx, Part 1.

of the bacilli into the shaven and epilated epidermis and afterwards covered the part with collodion, but any one of these procedures is sufficient to cause injury enough to remove the skin from the category of health.

The whole question is one of the seed and of the soil, and in the bulk of experimental work which has been done on this subject the seed alone, or practically alone, has been considered. It is no doubt very important to know if bacilli can make their way through an absolutely healthy mucosa, at least healthy so far as our methods of examination allow us to judge; but so frequently are diseased conditions of the bowel present that it is even of more consequence from a practical point of view to study the question under such unhealthy circumstances. More especially is this the case when one considers that it is just at the period of life when the bacilli are supposed to enter most frequently by this route, and in the class of society which pays the heaviest toll to tuberculosis, that the intestine is more frequently than not in a state of catarrh. It should be mentioned that Moro[24] and Ficker[25] found that hunger and exercise allowed of a more easy passage of organisms through the intestinal mucosa, but so far as I am aware no digestion experiments have been carried out with the intestine just recovering from, or in a state of, catarrh.

In view of the above facts — the contradictory results obtained by different workers; the faulty technic of many of the experiments; and the immense importance to prophylaxis — the problem seemed to me worthy of further consideration.

As previously mentioned, many have been the methods devised for the importation of the bacilli into the gastro-intestinal tract without causing at the same time insufflation: this is almost certain to occur, as my earlier work on the subject showed,[19] if one introduces emulsions of the bovine type of the organism through the stomach-tube, but is not nearly so likely, if the human and less pathogenic variety for rabbits is used. Unfortunately the majority of the other methods, especially those necessitating some abdominal operation, admit of additional fallacies, such as direct infection of the tissues or peritoneum, which readily ensues not only with the bovine but also with the human bacillus. It was thus early evident to me that no conclusions could be drawn from experiments carried out on these lines, and the following method suggested itself:

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24. Moro: Arch. f. Kinderh., xliii, 340.
25. Ficker: Arch. f. Hyg., liv, 354.
26. Fraser: Brit. Med. Jour., April 12, 1913.
27. Stiles: Seventeenth International Congress Med., Sec. x, London, 1913.

TECHNIC

The bacilli, suspended in oil, were placed in a gelatine capsule, which was slipped just within the nozzle of a stomach-tube (catheter No. 8), and ejected by forcing a stream of water through the tube after its passage. Immediately when the capsule comes in contact with mucus or any watery fluid it is dissolved and the bacilli make their escape. As this might happen before the capsule had reached the stomach through coming in contact with mucus in the mouth or pharynx, the capsule and stomach-tube were well coated with vaselin. Considerable experience with this method has shown me that insufflation can be avoided with the utmost certainty. In only one case out of fifty did inhalation tuberculosis occur, and in this instance too large a catheter had been used, and difficulty was experienced in passing it. Sufficient time had in this way been allowed for the coating of vaselin to be removed and for the mucus of the pharynx to dissolve the capsule. On withdrawing the tube a portion of the capsule with some of the tubercle culture attached was found still *in situ*, so that either during the introduction or withdrawal of the tube an opportunity for aspiration had been given.

In the present research both the bovine and human types of the bacillus have been experimented with, and their behavior in the normal and abnormal intestine studied. Since in the human subject, and especially during childhood, catarrhal states of the bowel are so common, it seemed to me that if one could induce a catarrh of the intestine the experimental conditions would more closely approximate to the natural situation. This I at first attempted to do by the administration of croton oil, castor oil and ground glass, but without success, although large doses of all these substances were given. Prof. Stockman, whom I consulted on this matter, suggested infusion of senna leaves and gamboge, and each of these substances I found quite efficient for the purpose. Of the first six animals to which infusion of senna was administered through the stomach-tube, five developed within a few hours a most profuse diarrhea, and died shortly afterwards. Examination after death revealed some injection of the intestines, and in a few cases hemorrhage into the wall of the cecum. As I found it difficult to guage the requisite dose of the infusion of senna, due I suppose to the fact that the infusion was not always of the same strength, and as the pharmaceutical product, cathartic acid prepared by Merck, was rather inert, I ultimately confined my attention to gamboge, and it was with this drug that enteritis was induced in most of the cases. I found that .75 gm. was a suitable dose for a full-grown rabbit, and .5 gm. for younger animals. As a rule those quantities set up a fairly profuse diarrhea with fluid stools containing mucus but no blood, from which the animals recovered in two or three days. Occasionally a rabbit was found somewhat immune to the drug, and no diarrhea or only a slight looseness of the bowels resulted, even after two or three successive doses of 0.75 gm. Some rabbits, on the other hand, were exceedingly susceptible, and with 0.5 gm. developed a most severe diarrhea, which terminated fatally within twenty-four hours. In my experience the rabbits which behaved in this latter fashion were those which possessed some wild blood. The tubercle bacilli were customarily administered one or two days after the cessation of the diarrhea, but in one case the inoculation was delayed as long as eight days.

To avoid as much as possible other means of infection than the one under consideration, cages improvised from orange boxes were used, and these were burned immediately after the completion of the experiment, so that no cage was employed for more than one animal; in addition the hutches were kept scrupulously clean.

Before describing in detail my own researches I might be permitted to mention how very important is the method by which a diagnosis of tuberculosis is made in any experimental work on rabbits and guinea-pigs. It is of course necessary to make certain that one is dealing with a true tuberculous lesion and not with pseudotuberculosis, and a sure diagnosis can only rest on a bacteriologic basis. Pseudotuberculosis is

not an uncommon disease, and has occurred in both my control and experimental animals. To the naked eye the lesions produced in this malady are very similar to these caused by Koch's bacillus, and they have a marked tendency to affect the very parts selected by the true tuberculous infection. It occurs in the form of nodules and caseous areas, but the nodules as a rule are whiter and lack the pearl-like centers, and the caseous foci have none of the yellowish tint possessed by those met with in true tuberculosis. Nevertheless, in the earlier stages and in the miliary forms it is impossible to differentiate between them except on the ground of the bacteriologic finding. Histologically true tuberculosis in animals has in my experience no characteristic picture (as in his human subject) and thus it is essential to make films of the different lesions and examine for the tubercle bacillus. In doubtful cases when acid-fast organisms are absent, films of the spleen, as was pointed out to me by Dr. Blair Martin, will usually disclose the presence of the pseudotubercle bacillus.

EXPERIMENTS WITH THE BOVINE TYPE OF THE TUBERCLE BACILLUS

A. Short-Period Experiments in Healthy Animals

In the series shown in Table 1 the animals were killed at short intervals, varying between four and ten days, after the introduction of the culture. This was exceedingly virulent (three weeks old) and, with one exception, when the dose was 10 mg., each capsule contained 5 mg. Emulsions of one lung and all the mesenteric glands from each case were made and introduced intraperitoneally through a small abdominal wound in rabbits, which were used for the secondary inoculations because it had been learned from previous work that guinea-pigs not infrequently died shortly after the injection of the emulsions; and in addition it was thought that rabbits, being very susceptible to the bovine type of the bacillus, would prove to be more delicate test animals.

Three of the animals inoculated with an emulsion of lung died too soon to give the disease time to develop, but in all the others ample time was allowed. In two of the animals inoculated with mesenteric glands, tuberculosis developed, thus showing that living tubercle bacilli had migrated to the mesenteric glands, but in not one instance were living bacilli demonstrated in the lungs by animal experiment. Both of the above mentioned infected animals had lived for a period of fifty-three days, and yet the disease was slight. It consisted of small tubercles situated chiefly in the omentum, but also in the mesentery and diaphragm, and, apparently spreading by the lymphatics, the disease had infected the pleural cavities, several small subpleural nodules being visible. Films of the nodules revealed very scanty tubercle bacilli.

Such a picture of tuberculosis caused by the bovine bacillus is most unusual after such a prolonged opportunity for growth — fifty-three

days. In one rabbit inoculated intraperitoneally with a very minute dose of the same culture as was used in the primary experiments, death occurred at the end of three days, when the peritoneum was found studded with minute tubercles containing abundant tubercle bacilli. And, moreover, in the inhalation experiments with the same type of organism, but another culture, the lungs were absolutely solid within thirty days.

TABLE 1.—Short-Period Experiments in Healthy Rabbits with Bovine Tubercle Bacilli in Gelatin Capsule Introduced Per Stomach-Tube

No.	Dose in Mg.	Duration of Experiment, Days	Method of Diagnosis: Rabbit Inoculated Intraperitoneally with	Duration of Secondary Inoculation, Days	Result
1	5	4	Mesenteric glands	55	Negative.
			Lung	55	Negative.
2	5	4	Mesenteric glands	19	Negative
			Lung	55	Negative.
3	5	6	Mesenteric glands	53	Slight peritonitis and few nodules in lungs; tubercle bacilli present but scanty.
			Lung	53	Negative.
4	5	6	Mesenteric glands	53	Slight peritonitis with numerous nodules in lungs and pleura, containing very scanty tubercle bacilli.
			Lungs	53	Negative.
5	5	6	Mesenteric glands	53	Negative.
			Lung	53	Negative.
6	5	8	Mesenteric glands	17	Negative.
7	5	8	Mesenteric glands	51	Negative.
			Lung	51	Negative.
8	5	8	Mesenteric glands	51	Negative.
			Lungs	51	Negative.
9	5	10	Mesenteric glands	49	Negative.
			Lung	10	Negative.
10	5	10	Mesenteric glands	20	Negative.
			Lung	49	Negative.
11	5	10	Mesenteric glands	20	Negative.
			Lung	2	Septic peritonitis.
12	10	8	Mesenteric glands	21	Pseudo tubercle.
			Lung	3	Septic peritonitis.

This very different picture would seem to show that tubercle bacilli in their passage through the intestinal mucosa diminish considerably in virulence, but, as previously mentioned, because tuberculosis developed when they were introduced into the peritoneum it does not necessarily follow that disease would ultimately have arisen in the glands themselves. Nevertheless the fact remains, that living tubercle bacilli can pass through the apparently healthy mucosa and reach the mesenteric glands within a period of six days, but such is not of very frequent occurrence.

B. Short-Period Experiments in Rabbits after Diarrhea

In the second series of short-period experiments (Table 2) the bacilli were introduced in greater quantity (10 mg. being used in all cases) after the animal had recovered from an intestinal catarrh induced by an infusion of senna leaves. In only one case out of five were living tubercle bacilli demonstrated in the mesenteric glands, but in not one instance were they shown to have reached the lungs within a matter of six days. One would have expected the catarrh to have favored the migration of the organisms through the wall of the gut, but experiment does not lend any support to such an idea.

TABLE 2.—SHORT-PERIOD EXPERIMENTS IN RABBITS AFTER DIARRHEA, WITH BOVINE TUBERCLE BACILLI IN GELATIN CAPSULE INTRODUCED PER STOMACH-TUBE*

No.	Dose in Mg.	Time After Diarrhea Ceased, Days	Duration of Experiment, Days	Method of Diagnosis: Rabbit Inoculated Intraperitoneally with	Duration of Secondary Experiment, Days	Result
1	10	1	6	Mesenteric glands	67	Tuberculosis.
				Lung	13	Negative.
2	10	1	6	Mesenteric glands	68	Negative.
				Lung	69	Negative.
3	10	1	6	Mesenteric glands	69	Negative.
				Lung	69	Negative.
4	10	2	8	Mesenteric glands	66	Negative.
				Lung	5	Negative.
5	10	2	8	Mesenteric glands	66	Negative.
				Lung	5	Septic peritonitis.

* In these animals diarrhea was induced by an infusion of senna.

C. Long-Period Experiments in Healthy Animals

In this series of so-called long-period experiments the animals were kept alive a sufficient time for the disease to develop. The dose of the culture varied between 10 and 20 mg. and was administered in a gelatin capsule with the stomach-tube. It is seen that the majority of the animals (87 per cent.) which lived forty-six days or longer developed tuberculosis. In all the positive cases the gastro-intestinal tract presented lesions visible to the naked eye, and, with one exception, where undoubtedly insufflation had occurred, the intestinal foci were the most extensive. The appendix and adenoid patch at the ileocecal valve invariably, and in the earliest cases these parts alone, were the seat of the mischief. In one experiment, however, of only forty-six days' duration the cecum was in addition studded throughout with small nodules, and the mesenteric glands were extensively involved, presenting numer-

TABLE 3.—Long-Period Digestion Experiments in Healthy Rabbits with Bovine Tubercle Bacilli in Gelatin Capsule Introduced Per stomach-Tube

No.	Dose in Mg.	Duration in Days	Result
1	15	26	*Young rabbit, under 3 months.* This animal in some way fractured its spine and was killed rather soon. Absolutely no evidence of tuberculosis anywhere.
2	20	30	*Full-grown rabbit. Died.* Lobar pneumonia apparently due to a plague-like organism. Absolutely no evidence of tuberculosis anywhere.
3	15	32	*Young rabbit.* No tuberculosis discovered anywhere.
4	20	35	*Medium-sized rabbit.* Miliary tuberculosis in intestine, spleen, lungs, and mesenteric glands. No tubercle bacilli discovered, but pseudo-tubercle bacilli.
5	10–15	46	*Medium-sized rabbit.* Appendix and ileo-cecal valve region slightly affected. One or two caseous foci in mesenteric glands. No other lesions. Tubercle bacilli present.
6	10–15	46	*Full-grown brown rabbit.* Appendix and ileo-cecal valve region show numerous nodules; cecum studded all over with small nodules. Mesenteric glands enlarged and caseous. Lungs, spleen, and kidneys free. Tubercle bacilli present.
7	20	50	*Full-grown rabbit.* One or two nodules situated in appendix. Mesenteric glands and cecum free. In lungs a very extensive lesion, but at time of passing tube some difficulty was experienced, and in all probability insufflation occurred. No tubercle bacilli in mesenteric glands, but abundant in lungs and scanty in appendix nodules.
8	20	50	*Full-grown rabbit.* Two or three nodules in appendix and one in adenoid patch at ileo-cecal valve, with questionable focus in mesenteric glands. Lungs marked by anthracosis é pigmentation of bronchial glands, but no tuberculosis. Films from nodule in appendix, cecum, and mesenteric glands show scanty tubercle bacilli.
9	20	54	*Medium-sized rabbit.* One or two nodules at ileo-cecal valve; one or two Peyer's patches show foci of caseation and ulceration; caseous foci in mesenteric glands and spleen. The nodules are whiter in color than usual tubercular lesions, and more uniform in consistence. Lungs anthracotic, but free from any nodules. No tubercle bacilli discovered, but in spleen and mesenteric glands pseudo-tubercle bacilli were present.
10	20	70	*Medium-sized rabbit.* Appendix and cecum at ileo-cecal valve seat of mischief; several Peyer's patches infected. Mesenteric glands show a very minute caseous focus, and in upper lobe of right lung is a small nodule. Lungs markedly anthracotic. Very scanty tubercle bacilli present in appendix and mesenteric gland nodules.
11	20	74	*Full-grown rabbit.* Moderate degree of tuberculosis in appendix and ileo-cecal valve regions. Four Peyer's patches involved. Mesenteric glands, spleen, and kidneys free. One very small nodule in left lung. Tubercle bacilli present in cecal nodules.
12	20	84	*Medium-sized rabbit.* Appendix and ileo-cecal valve regions slightly affected, but numerous foci in lower part of small intestine, with extensive infection of several Peyer's patches. In connection with latter are several caseous lymph-nodes in mesentery. Chief group of mesenteric glands caseous. One nodule in lung. Tubercle bacilli present.
13	20	100	*Full-grown rabbit.* Appendix fairly extensively infected, the nodules varying in size from pin-head to split pea, but ileo-cecal valve region only shows one or two nodules. Mesenteric glands contain one or two caseous foci. Two Peyer's patches involved slightly. Lungs throughout are seat of tubercular foci varying in size from pin-head to pea. Tubercle bacilli present, but scanty.

ous caseous foci. In the majority of the positive experiments the mesenteric glands were also affected, and in those rabbits which had survived fifty days or longer infection of the Peyer's patches was found, and in those killed seventy days or more after inoculation an occasional nodule was encountered in the lungs. In no case was the spleen observed to be the seat of tuberculosis.

These experiments prove that rabbits can be infected by feeding with tuberculous material. In every instance the most extensive mischief was situated locally in the intestine, and thus they lend absolutely no support to the idea that primary pulmonary tuberculosis, or indeed primary tuberculosis of any organ other than the bowel, can arise from bacilli which have gained admission by the intestinal route.

D. Long-Period Experiments in Rabbits after Diarrhea

From Table 4 it will be observed that of the seventeen animals which lived longer than nineteen days, fourteen, that is, 82 per cent., developed tuberculosis, and all those that survived at least thirty days, that is, 100 per cent., showed definite infection of the gastro-intestinal tract. As in the healthy animals the earliest lesions were situated in the appendix and lymphoid area at the ileocecal valve in the form of small whitish nodules about the size of a pinhead lying immediately under the peritoneal coat. When the animals had lived for at least forty-one days the mesenteric glands invariably presented caseous foci, and the cecum and lungs were as a rule the seat of tuberculosis. In its earliest stages the mischief in the cecum was evidenced by small nodules situated on the wall of the bowel or on the free edges of the valvulae conniventes, simulating very closely tuberculosis of the vocal cords. In the later stages disintegration of the foci had occurred, with the formation of ulcers, which in the experiments of seventy-three days' duration had a diameter of 12 cm. The lungs were variously affected; in one case of forty-five days' duration numerous nodules were scattered throughout both lungs; in one of fifty-six days only a few nodules were present, while in another of sixty days the lungs were free of tuberculous mischief, but in all those that had survived sixty-five days the lungs were extensively infected. In four of the animals which survived at least forty-five days the spleen was the seat of definite nodules. In the experiments of forty-five, sixty and seventy-three days, respectively, tuberculous ulceration of the stomach had taken place.

It is thus seen that in the animals which had previously suffered from enteritis the lesions were on the whole similarly distributed to those occurring in rabbits not so affected. Not only, however, did they occur with greater frequency in the former, but they made their appearance at a much earlier date, in one case as soon as nineteen

TABLE 4.—Long-Period Digestion Experiments in Rabbits, After Diarrhea, with Bovine Tubercle Bacilli in Gelatin Capsule Introduced Per Stomach-Tube. In all the Animals Except Those Marked by Asterisk the Diarrhea was Induced by Gamboge; in the Others Infusion of Senna was Used

No.	Dose, mg.	Time After Diarrhea in Days	Duration in Days	Result
1	15	..	11	Negative. No tuberculosis anywhere.
2	5–10	0	14	*Medium-sized rabbit.* Two small nodules present in small intestine with a small caseous focus in mesenteric glands. No tubercle bacilli found. Lungs, spleen and kidneys free.
3	10–15	1	19	*Medium-sized rabbit.* One or two nodules in adenoid patch at ileo-cecal valve and also in appendix. Mesenteric glands and lungs free. Tubercle bacilli present.
4	5–10	0	20	*Full-grown rabbit.* Absolutely no evidence of tuberculosis anywhere.
5	5–10	0	25	*Young rabbit.* In appendix were some suspicious nodules, but films failed to reveal the presence of tubercle bacilli.
6	10–15	?	25	*Young rabbit about 3 months old.* This animal had suffered from only a very slight diarrhea. Extensive pseudo-tuberculosis of intestine, but no true tubercular lesions anywhere.
7	10–15	4	30	*Medium-sized rabbit.* Some small nodules in wall of appendix and in adenoid patch at ileo-cecal valve. No other lesions. Tubercle bacilli present.
8	10–15	2	41	*Young rabbit.* Nodules in appendix, small intestine at ileo-cecal valve, on valvulae conniventes in cecum; mesenteric glands caseous; tuberculosis of upper lobes of both lungs. Spleen, kidney and liver free. Tubercle bacilli present.
9	10–15	2	44	*Medium-sized rabbit.* Nodules in appendix; mesenteric glands caseous; extensive mischief in lungs (no suspicion of insufflation). Tubercle bacilli present.
10	15	1	45	*Medium-sized rabbit.* Nodules in appendix and small intestine at ileo-cecal valve (very abundant in both regions). Valvulae conniventes here and there infected. Mesenteric glands enlarged and caseous. Ulcer in stomach. Numerous small nodules scattered throughout both lungs. Spleen seat of tuberculosis. Tubercle bacilli present.
11	15	1	45	*Medium-sized rabbit.* Nodules in appendix and small intestine, at ileo-cecal valve containing tubercle bacilli. Questionable focus in mesenteric glands, but no tubercle bacilli present. Lungs and spleen free.
12	10–15	2	56	*Full-grown rabbit.* Extensive tuberculosis in appendix, in adenoid patch at ileo-cecal valve, in cecum, Peyer's patches, and mesenteric glands. One or two nodules situated in lungs. Tubercle bacilli present.
13	15	1	60	*Full-grown rabbit.* Appendix and ileo-cecal valve region riddled with nodules. Ulcer on posterior wall of stomach. Mesenteric glands slightly affected. Lungs, spleen and kidneys free. Tubercle bacilli present in nodules in small intestine, in wall of gastric ulcer, and in lesions in mesenteric glands.
14	15	2	60	*Full-grown rabbit.* Appendix and small intestine at entrance to cecum riddled with nodules; ulcers present on valvulae conniventes and wall of cecum; mesenteric glands infected. Lungs and spleen healthy. Tubercle bacilli present in lesions in appendix and mesenteric glands.

TABLE 4.—CONTINUED

No.	Dose, mg.	Time After Diarrhea in Days	Duration in Days	Result
15	2–3	4	63	*Medium-sized rabbit.* Appendix and small intestine at ileocecal valve are seat of numerous nodules. Many Peyer's patches infected. Ulcers on wall of cecum and valvulae conniventes. Mesenteric glands caseous. Numerous nodules in lungs, some large and some small. Kidneys, spleen and liver healthy. Tubercle bacilli in lesions in mesenteric glands and lungs.
16	2–3	4	70	*Medium-sized rabbit.* Appendix and small intestine at entrance to cecum show very many nodules. Solitary nodule on one of valvulae conniventes and one Peyer's patch infected. Mesenteric glands, lungs (especially the left), bronchial glands, and spleen are also seat of tubercular disease. Tubercle bacilli present.
*17	10	2	73	*Medium-sized rabbit.* Appendix, cecum and lower part of small intestine extensively diseased. Mesenteric glands caseous. Spleen contains nodules of tuberculosis as also both lungs. Tubercle bacilli present.
*18	10	2	73	*Medium-sized rabbit.* Appendix almost entirely caseous, cecum contains many nodules, and mesenteric glands caseous. Two large ulcers with caseous floors in stomach. One or two nodules in lungs. Spleen and kidneys healthy. Tubercle bacilli present in all lesions.
*19	10	2	73	*Medium-sized rabbit.* Very extensive infection of cecum, where valvulae conniventes are studded with nodules and ulcers are present on wall. Small intestine and appendix also extensively infected. Mesenteric glands caseous, spleen the seat of nodules and caseous foci. Both lungs are riddled with nodules varying in size from that of a pin-head to that of a split pea. Lesions show presence of tubercle bacilli.

days after the inoculation, and they ran a much more rapid course and were disseminated more widely. Whereas in the healthy animals which survived seventy-four days the disease was evidenced only by a few nodules in the appendix and adenoid patch at the ileocecal valve, with some caseation of the mesenteric glands and an occasional nodule in the lungs, in the others, that is, the animals recovering from enteritis, and that with a dose of only one-half to one-tenth of the amount, the appendix and ileocecal valve were extensively infiltrated, the valvulae conniventes and wall of the cecum showed numerous nodules with ulceration, the Peyer's patches and mesenteric glands were enlarged and caseous, and the lungs invariably, and the spleen almost constantly and extensively, involved. These points are very well illustrated in Tables 5 and 6, showing the comparative extent of the lesions in the two series of cases.

It would thus seem undoubted that a catarrhal condition of the intestine favors an earlier and more severe infection. This does not

TABLE 5.—Showing Comparative Extent of Lesions at Different Parts in Series of Healthy Rabbits (Table 3). In all Lesions Tubercle Bacilli Were Scanty

No.	Dose in Mg.	Duration in Days	Appendix	Ileocecal Valve	Cecum	Peyer's Patch	Mesenteric Glands	Lung	Spleen	Stomach	
1	15	26	—	—	—	—	—	—	—	—	
2	20	30	—	—	—	—	—	—	—	—	
3	15	32	—	—	—	—	—	—	—	—	
4	20	35	—	—	—	—	—	—	—	—	Pseudo-tuber-culosis
5	20	46	+	+	—	—	+	—	—	—	
6	20	46	+++	+++	+++	—	+++	—	—	—	
7	20	50	+	—	—	—	—	Extensive	—	—	Insuffla-tion
8	20	50	+	+	—	—	?	—	—	—	
9	20	54	—	—	—	—	—	—	—	—	Pseudo-tuber-culosis
10	20	70	+	+	—	++	+	+	—	—	
11	20	74	+	—	—	+	+	—	—	—	
12	20	84	+	+	—	++ ++	+++	+	—	—	
13	20	100	+++	+	—	++	++	++ ++	—	—	

TABLE 6.—Showing Comparative Extent of Lesions at Different Parts in Series of Diarrheal Animals (Table 4). In all Lesions Examined Tubercle Bacilli Were Abundant

No.	Dose in Mg.	Duration in Days	Appendix	Ileocecal Valve	Cecum	Peyer's Patch	Mesenteric Glands	Lung	Spleen	Stomach
1	15	11	—	—	—	—	—	—	—	—
2	5–10	14	—	?	—	—	?	—	—	—
3	10–15	19	+	+	—	—	—	—	—	—
4	5–10	20	—	—	—	—	—	—	—	—
5	5–10	25	?	—	—	—	—	—	—	—
6	10–15	25	—	—	—	—	—	—	—	—
7	10–15	30	+	+	—	—	—	—	—	—
8	10–15	41	+	+	+	—	+	+	—	—
9	10–15	44	+	+	—	—	+	+++	—	—
10	15	45	++	+++	+	—	+++	+	+	+
11	15	45	+	+	—	—	—	—	—	—
12	10–15	56	+++	++	++	+	+	+	—	—
13	15	60	+++	+++	—	—	+	—	—	+
14	15	60	+++	+++	++	—	+	—	—	—
15	2–3	63	+++	+++	++	+++	++	++	—	—
16	2–3	70	+++	+++	+	+	++	++	+	—
17	10	73	+++	+++	+++	+++	++	++	+	—
18	10	73	++++	+++	++	—	++	+	—	+
19	10	73	++++	+++++	+++	+++	++	+++	+	—

result, however, from the mucosa becoming more pervious to the passage of organisms, but rather by it being made a more ready prey to the disease. The lesions developing in the weakened tissue are more numerous, progress more rapidly and allow of a more easy dissemination from the greater abundance of the proliferating tubercle bacilli present.

EXPERIMENTS WITH THE HUMAN TYPE OF THE TUBERCLE BACILLUS

In the series shown in Table 7 the bacilli were suspended in milk and introduced through the stomach-tube after twenty-four hours' fasting. With two exceptions, when three and seven doses were administered, each animal received the emulsion on only one occasion. The animals were kept alive for from forty-one to fifty days and yet in

TABLE 7.—EMULSION OF HUMAN TUBERCLE BACILLI ADMINISTERED TO RABBITS BY STOMACH-TUBE AFTER TWENTY-FOUR HOURS' STARVATION

No.	Dose, Mg.	No. of Doses	Duration of Experiment, Days	Result
1	240	3	50	Negative
2	480	1	43	Negative
3	480	7	49	Negative
4	840	1	41	Negative
5	840	1	46	Negative
6	1,200	1	49	Negative
7	1,200	1	45	Negative
8	2,400	1	46	Negative
9	2,400	1	43	Negative

not one instance was any disease visible to the naked eye, nor were bacilli discovered in films of the mesenteric glands. It must be admitted, however, that the quantity of the infective material administered was small, especially when compared with that used in the experiments with the more pathogenic variety of the bacillus.

In another series (Table 8) of healthy rabbits human sputum containing very numerous tubercle bacilli was the infecting agent used. As in the preceding experiments it was administered by the stomach-tube, and in all but a single case on more than one occasion. This probably accounts for the frequency with which insufflation tuberculosis was encountered. The animals were allowed to live for periods varying from eighteen to fifty-six days. In five of nine cases so treated there was no tuberculosis anywhere; in three (33 per cent.) the lungs were infected, but undoubtedly as the result of insufflation; and in two (one with, and one without, pulmonary mischief) tubercle bacilli were detected in films of the mesenteric glands; but in the whole series there was not one example of manifest tuberculosis of the abdominal cavity.

In view of the low degree of virulence for the rabbit of this variety of the tubercle bacillus, some digestion experiments were conducted in healthy animals with much larger doses (Table 9). The bacilli, as in the investigations with the bovine type of the organism, were enclosed in gelatin capsules and introduced by means of the stomach-tube. In

TABLE 8.—HUMAN TUBERCULAR SPUTUM ADMINISTERED TO RABBITS BY THE STOMACH-TUBE

No.	Dose oz.	No. of Doses	Duration of Experiment, Days	Result
1	1	6	50	Insufflation tuberculosis. No tuberculosis in mesenteric glands.
2	1	3	46	Negative.
3	1	6	50	Negative.
4	½	5	48	Tubercle bacilli in films of mesenteric glands.
5	½	5	48	Insufflation tuberculosis. Tubercle bacilli in films of mesenteric glands.
6	1	3	49	Negative.
7	1	1	54	Negative.
8	1	3	18	Negative.
9	1	3	56	Insufflation tuberculosis in lungs. No tubercle bacilli in films of mesenteric glands.

TABLE 9.—LONG-PERIOD DIGESTION EXPERIMENTS IN HEALTHY RABBITS WITH HUMAN TUBERCLE BACILLI

No.	Dose in Mg.	Duration in Days	Result
1	20	96	Four small foci in adenoid patch at ileo-cecal valve containing numerous tubercle bacilli. Several nodules in left lung with scanty bacilli. Appendix, mesenteric glands and spleen free.
2	20	117	Several nodules in left lung with fairly abundant tubercle bacilli. Questionable focus in adenoid patch at ileo-cecal valve and also in mesenteric glands, but films did not reveal any tubercle bacilli.

all, four animals were so treated, but unfortunately two died before sufficient time had been allowed for the disease to develop. The remaining two animals, however, were permitted to survive for ninety-six and 117 days, and in both instances they presented definite tuberculous lesions. The mischief, as one can easily understand, was much less extensive than in the experiments with the more virulent type of organism. In the ninety-six day experiment a few nodules containing numerous tubercle bacilli were present in the adenoid patch at the ileocecal

valve, with, in addition, several foci about the size of a split-pea in the left lung. In the animal which survived 117 days the only definite tuberculous lesions were found in the left lung; in the adenoid patch at the ileocecal valve a questionable pin-point-sized focus was observed, but examination of films failed to reveal any tubercle bacilli. It would thus seem that here we had to deal with a case of true primary pulmonary tuberculosis arising from bacilli which had gained admission to the economy by the alimentary tract. I do not think that in this case there need be any question of insufflation, considering the slight lesion after such a prolonged period — 117 days. In my previous work on inhalation tuberculosis the pulmonary lesions were very extensive within sixty days, and the same was true of insufflation tuberculosis occurring in the digestion experiments carried out before the technic had been improved as described above.

TABLE 10.—LONG-PERIOD EXPERIMENTS IN RABBITS WITH HUMAN-TUBERCLE BACILLI WHICH WERE INTRODUCED IN GELATIN CAPSULES PER STOMACH-TUBE AFTER DIARRHEA. IN THESE ANIMALS THE DIARRHEA WAS INDUCED BY GAMBOGE

No.	Dose, mg.	Time After Diarrhea Ceased, Days	Duration of Experiment, Days	Result
1	15–20	2	37	*Full-grown rabbit.* One small nodule situated in adenoid tissue in small intestine at entrance to cecum. Tubercle bacilli found in films of nodule.
2	15–20	2	49	*Medium-sized rabbit.* Two small nodules in appendix and three in small intestine at entrance to cecum. Mesenteric glands extensively caseous. Lungs free. Tubercle bacilli present.
3	15–20	2	55	*Full-grown rabbit.* Nodule in appendix with a caseous focus in mesenteric glands. Lungs, spleen, and kidney free. Tubercle bacilli present.
4	15–20	8	61	*Full-grown rabbit.* No evidence of tuberculosis anywhere.
5	15–20	2	63	*Medium-sized rabbit.* Two small nodules in small intestine and mesenteric glands caseous. Lungs and spleen free. Tubercle bacilli present.

In a fourth series (Table 10), comprising five animals, enteritis was induced by the administration of gamboge. The bacilli (15 to 20 mg. of a three-weeks-old culture) were enclosed as before in a gelatin capsule and introduced by means of the stomach tube, in four of the animals two days, and in the other eight days, after the cessation of the diarrhea. With the exception of the rabbit that had recovered from the diarrhea eight days previous to the inoculation, all showed evidence of tuberculosis. As in the experiments with the bovine tubercle bacillus, the appendix and adenoid patch at the ileocecal valve were the seats of election, so far as the alimentary canal was concerned, although

in one case the small intestine alone was affected. In all only an occasional nodule about the size of a pinhead was visible in the intestinal tract, and in one experiment of thirty-seven days such was the whole extent of the disease. In the other three positive cases which lived forty-nine, fifty-five and sixty-three days, respectively, the mesenteric glands were also infected, and usually more extensively than the intestine itself. In no instance did either the lung or spleen present lesions visible to the naked eye, which was due of course to the fact that the animals had not been allowed to live long enough.

These results with the human type of the bacillus are in striking contrast to what prevailed with the bovine, in which an intestinal lesion was not only constantly present, but was invariably the most severe. With the human type of the organism, on the other hand, an intestinal lesion did not always result, and when present was often less extensive than the mischief in the internal organs, e. g., the mesenteric glands in the diarrheal animals, and the lungs in the non-diarrheal animals. This may in part be accounted for by the circumstance that not only do animals of a different species, but also the different organs of the same animal, vary in their susceptibility to any individual organism. It is just possible, however, that in every case there had been an intestinal lesion, but being the least severe it had healed in the experiment of longest duration — 117 days — and all trace of the portal of entry had been lost. This, as will be remembered, was suggested by Heller, and such a termination to the primary intestinal focus is not unlikely in view of the well-known fact that lesions in the rabbit due to the human bacillus after reaching a certain stage incline to be retrogressive.

If one explains the absence of an intestinal lesion in this way, then my experiments lend no support to the idea that true primary pulmonary tuberculosis can arise from the ingestion of the tubercle bacillus. To my mind such a reading of the findings is most probably correct, although if the intestinal lesion ultimately heals, while the focus in a distant organ extends, then, at certain stages of the infection, one may apparently have to deal with a primary lesion in the lung, or some other distant organ, from bacilli which have entered by the bowel. This is of course a point of considerable importance in comparing our experimental results with the disease as it occurs in man, for in all probability the process has at the death of the patient been in existence for a prolonged period, and ample time has been given for the disappearance of the local mischief at the point of entry.

In consequence of the wide distribution of bovine lesions in childhood, as conclusively shown by the work of the British Royal Commission, Fraser[26] and Stiles[27] of Edinburgh and others, and the prevailing opinion that it is by the intestine that the bacillus which causes

this variety of the disease, chiefly, if not entirely, gains admission to the body, the experiments with the human type of the organism are specially interesting. In drawing analogies, however, one must surmise — and it is merely a surmise — that man is as little susceptible to the bovine as the rabbit is to the human bacillus. And if one premises this it would then seem possible to understand how the ingested bovine bacillus, without betraying its means of ingress, could gain entry to the economy in sufficient numbers to give rise to lesions. A primary intestinal lesion may, so far as we can see, be entirely absent, or if present, so slight that it might be overlooked, and yet a more extensive focus be present in the mesenteric glands or lungs, and in all probability in the bones also, though of this one has no experimental proof. For such an event, however, it is not improbable that a large dose of virulent bacilli is necessary so long as the intestine is in a healthy state, but so soon as the defensive powers of the bowel are weakened, even apparently to a very slight extent by a passing catarrh, as the above experiments very strikingly demonstrate, the possibility of such a contingency is very greatly enhanced. And, until the complete eradication of tuberculosis in cattle has been accomplished, the obvious lesson for us is, that our chief means of combating the spread of this variety of the disease consists in attention to the proper feeding of infants and children. All milk which is not above suspicion should be sterilized; everything likely to cause intestinal catarrh avoided, and no attack of diarrhea neglected.

CONCLUSIONS

1. Healthy rabbits can be infected by the ingestion of large amounts of bovine tubercle bacilli.

2. The bacilli can pass through the apparently healthy intestinal mucous membrane and reach the mesenteric glands within a period of six days: this, however, does not frequently happen.

3. When infection occurs the intestine is invariably the seat of lesions, and thus tuberculosis of any organ, other than the intestine, when due to the bovine tubercle bacillus, in the rabbit is always a secondary infection.

4. Catarrh of the intestine does not favor the absorption of the tubercle bacillus directly, but allows with a much smaller dose of a more constant, and also of an earlier and more widespread, infection of the bowel itself, and in this way facilitates dissemination.

5. Healthy rabbits may be infected by the ingestion of large amounts of the human tubercle bacillus.

6. Rabbits just recovered from intestinal catarrh constantly, and within a much shorter period, develop tuberculosis after the ingestion of human tubercle bacilli.

7. With the human type of organism a local intestinal lesion is not always present, and when present is slight in comparison with the diseased foci in the mesenteric glands or lungs.

8. Apparently primary pulmonary tuberculosis can be caused in rabbits by the ingestion of large amounts of human tubercle bacilli.

9. It would seem that the best means of combating the spread of bovine infection in childhood is in the avoidance of everything likely to cause intestinal catarrh, and in the sterilization of milk intended for the use of infants and children.

CONGENITAL ELEVATION OF THE SCAPULA (SPRENGEL'S DEFORMITY) OF FAMILIAL TYPE *

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Although a considerable number of instances of congenital unilateral elevation of the scapula and some fourteen of bilateral elevation have been reported in the literature since the affection was first described, the etiology of Sprengel's deformity remains obscure. Several theories have been advanced to explain the causation of the deformity, but without entering into them in any detail at this point, it may be briefly stated that none of them has proven satisfactory. It is chiefly with the object of throwing a new light on the etiology of Sprengel's deformity that I shall present the unique example of several instances of the affection, both unilateral and bilateral, all occurring in different members of one family.

An incomplete report of this family has already been made.[1] The branch of the family missing in that report (comprising numbers 3, 12, 13, 14 and 15 in the diagram, Fig. 1) was subsequently examined so that a complete presentation is now possible. Of the elder branches of the family (numbers 1, 2, 3 and 4), two were instances of bilateral congenital elevation of the scapula; of their fifteen children, seven presented the unilateral and two the bilateral deformity.

The diagram (Fig. 1) will schematically illustrate the family tree, indicating those suffering from the unilateral, those bilaterally affected, and those free from the deformity.[2]

The findings in each case will now be described in detail and some general considerations, based on the material described, will then be presented.

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* Presented in part before the Orthopedic Section of the New York Academy of Medicine, April, 1912.

* From the Surgical Department of the Mount Sinai Hospital Dispensary.

1. Neuhof, H.: Ztschr. f. Orthoped. Chirurg., 1913, xxxi, 519.

2. The members of the family tree are arranged in order of increasing age from left to right. The numbers correspond to the numbers in the detailed description of each member of the family. The scapular deformity is the only one noted in the diagram; confusion would have resulted if all the additional anomalies were here recorded. It is unfortunate that thorough examinations were not permitted by several of the affected individuals.

I. BILATERAL CONGENITAL ELEVATION OF THE SCAPULAE

CASE 1.—Herman H., aged 38. With the exception of "nervous dyspepsia" from which he suffered for many years, he has always been well, although never strong. He has never been able to abduct his arms beyond the horizontal without swinging them upwards, but has always been able to hold them above his head after they have been swung to that position. Active use of the upper extremities has always been difficult.

The individual is above the average in height and of spare build. His face is strikingly asymmetrical, the left eye and ear being fully 1.5 cm. higher than the right (Fig. 2). The thorax is very narrow and quite cylindrical. Both shoulder girdles seem almost detached from the chest-wall. The scapulae (Fig. 3) are displaced upwards, are far removed from the vertebral column, and are tilted forward to such a degree that they appear nearly horizontal in position. The right scapula is higher than the left. The detailed examination follows:

The *right scapula* is 17.5 cm. long and 16 cm. wide, the scapular index[3] thus being 72.14. The superior angle lies 4 cm. above the upper border of the clavicle; it is prolongated and pointed at the tip, making a sharp, visible and palpable

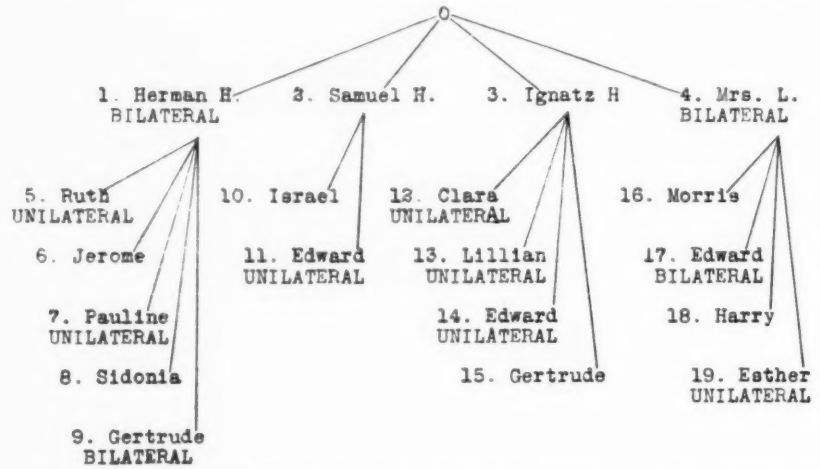

Fig. 1.—Genealogical chart showing congenital elevation of the scapula in author's cases.

spur in the supraclavicular fossa. This prolongation of the superior angle is largely cartilaginous, for only a small part of it is visible in the radiograph (Fig. 4). The supraspinata measures 6 cm., the infraspinata, 11.5 cm. The former is incurved on the latter, the resulting subscapular angle being about 110 degrees.[4] The scapula is tilted forward to an angle of 60 degrees with the vertical, making its inferior part very prominent. As above indicated, the scapula lies at a considerable distance from the median line — 10 cm. at its superior angle, 11 cm. at the scapular spine, and 12.5 cm. at the lower angle. Concerning the musculature of this bone, the levator anguli scapulae seems normal, but both the serratus

3. The normal scapular index $\frac{\text{breadth} \times 100}{\text{length}}$ for the European is 63.5, according to Dwight (based on the most extensive statistics).

4. The supraspinata is that portion of the bone lying above the spine of the scapula; the infraspinata is the portion below the scapular spine. The normal subscapular angle is about 130 degrees. (Quain.)

magnus and the trapezius (the latter chiefly in its lower half) are deficient to a marked extent. The scapular wing can be lifted well away from the thorax, the rhomboid muscles appearing to be the only ones left to hold it in place. The right arm can be actively abducted only to an angle of 50 degrees. The arm must be *swung* upwards to further elevate it. The scapula moves as soon as abduction begins and when the arm is carried to its full abduction of 50 degrees, the scapula is almost detached from the chest wall.

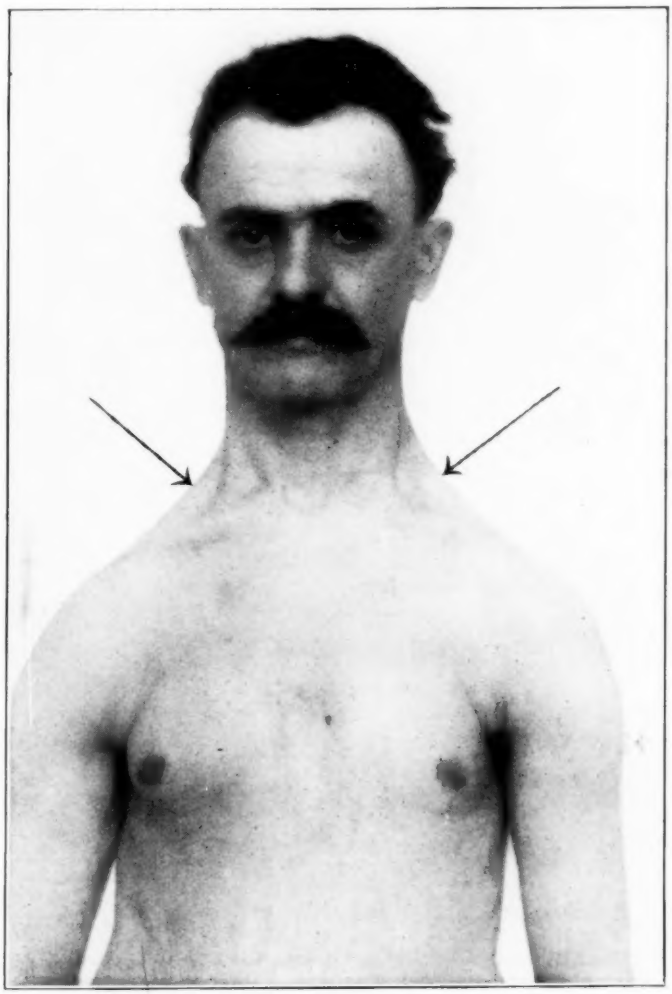

Fig. 2.—(Case 1) Herman H., front view; arrows point to the upper angles of the scapulae.

The peculiarities of the *left scapula* closely resemble those of the right, but are different in some details. It, too, is much shorter than normal, the length being 16.25 cm. and width 14.75 cm. (the scapular index is therefore 81.08). Although abnormally elevated, the left scapula is 2.5 cm. lower than the right (Figs. 2 and 3). Its superior angle is more flat and triangular in shape and

is very extensively cartilaginous; the prolongation of the angle is even more prominent in the supraclavicular fossa than on the right side, owing to a more extreme forward tilting of the left scapula (65 degrees with the vertical). The tip of the spur is 1.5 cm. above the clavicle. The supraspinata measures 6 cm. and the infraspinata, 10.25 cm.; the former is bent on the latter as on the opposite side, making about the same subscapular angle. The left scapula is further from the median line than the right—at its superior angle 11 cm., at the scapular

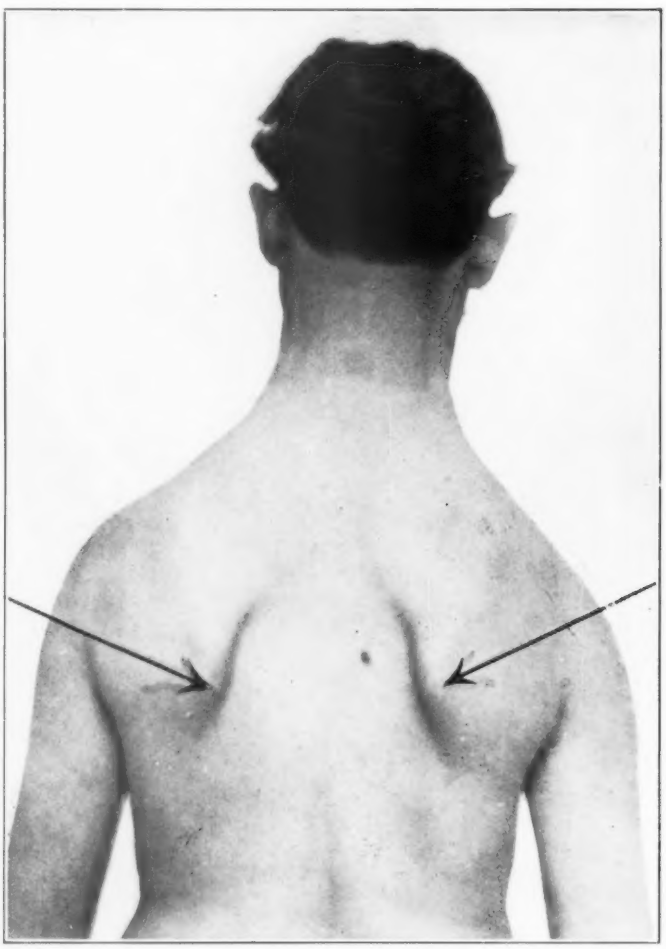

Fig. 3.—(Case 1) Herman H., rear view; arrows point to the inferior scapular angles.

spine 13 cm., at the inferior angle 14 cm. The muscular deficiencies, the limitation of abduction, and the migration of the scapula in abduction, are the same as on the right side.

In addition to the deformity of the shoulder-blades, several other abnormalties are to be noted. The strikingly narrow and barrel-shaped chest of this individual has already been mentioned. The circumferences at the nipples and at the lower thoracic aperture are both 75 cm. The ribs slant sharply downwards, especially

on the right side, and the intercostal spaces are irregular in width. The first ribs are spade-like, and flat at their anterior ends. As shown in the radiograph (Fig. 4), the last cervical vertebra is shortened in the vertical axis; the first dorsal vertebra is really situated in the lower cervical region.[5] There is, however, no vertebral deficiency; this was proven by a series of radiographs of the entire spinal column. The spine presents a right lateral scoliosis in the lower cervical region (not well seen in the radiograph or photographs) and a slight curvature in the opposite direction in the upper dorsal region.

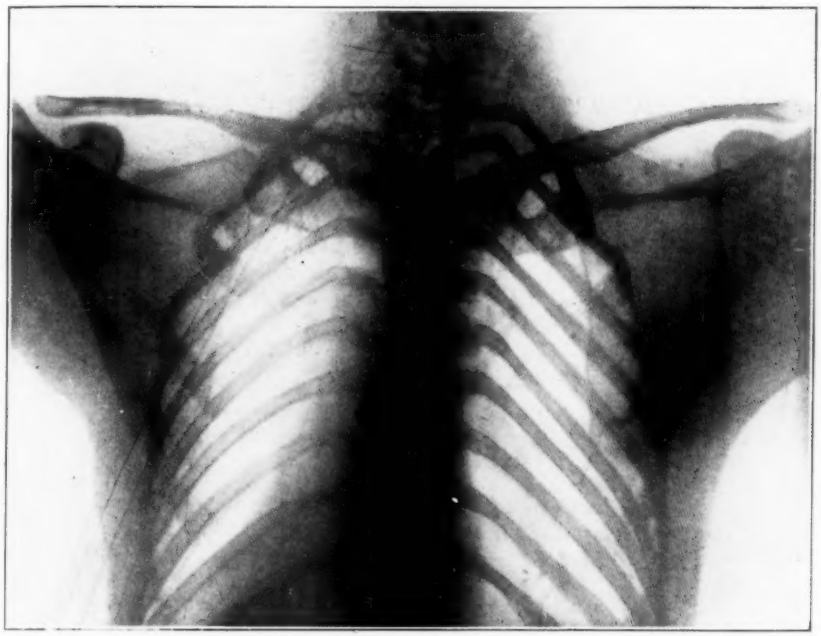

Fig. 4.—(Case 1) Herman H. Radiograph of the scapulae and thorax.

This patient presents no bony or muscular abnormalities other than those above described. To tabulate and summarize the findings in his case:

	Right Scapula	Left Scapula
Measurements	17.5 by 16 cm.	16.25 by 14.75 cm.
Index	72.14	81.08
Superior scapular angle..	4 cm. above clavicle; pointed; hook-like.	1.5 cm. above clavicle; blunter; more prominent.
Supraspinata	6 cm.	6 cm.
Infraspinata	11.5 cm.	10.25 cm.
Vertebral border of scapula from mid-line at:		
Superior angle	10 cm.	11 cm.
Spine of scapula....	11 cm.	13 cm.
Inferior angle	12.5 cm.	14 cm.

5. Measurement of the elevation of the scapula on the vertebral column as a standard, a method very generally employed, would therefore be very inaccurate in this instance.

	Right Scapula	Left Scapula
Rotation on frontal axis	60°	65°
Serratus magnus	Markedly deficient.	Markedly deficient
Trapezius	Markedly deficient.	Markedly deficient
Abduction of arms.....	To 50°	To 50°

The other features of interest are: Facial asymmetry; isolated cervical scoliosis; narrow last cervical vertebra; cervical position of first dorsal vertebra; very narrow, cylindrical thorax; obliquity of ribs; uneven intercostal spaces.

II. CONGENITAL (?) SCOLIOSIS

CASE 2.—Samuel H., aged 40. Always strong and healthy. He was first seriously impressed by the deformity of his spine at the age of 16, but believes it may have been present before that time.

Physical examination reveals a robust individual with well-developed musculature. There is a tremendous right lateral curvature of the dorsal spine and a correspondingly great bulging of the right thoracic wall in its posterior half. The lumbar spine presents a compensatory left lateral deviation. The movements of the spine are only slightly restricted. The right scapula is somewhat higher than the left,[6] but in size, shape and musculature, both scapulae are normal. No other abnormalities are to be found in this patient.

III. EXTENSIVE MUSCLE DEFECTS. DEFORMITY OF THE RIGHT SCAPULA. NO SPRENGEL

CASE 3.—Ignatz H., 43 years old. He has noticed that the right upper arm is much smaller than the left, and that this condition has been present for many years. Both arms, however, have always been useful for active work.

Physical examination reveals a thick set, very muscular individual, whose thorax appears very broad as viewed from behind.[7] The circumference of the thorax, however, is 91.5 cm. at the nipple level, and the right and left halves are practically equal in size. The apparent broadening of the chest is due to the great distance of the scapulae from the median line. Both bones are equidistant from that line — 13 cm. at the superior angles and 12.5 cm. at the inferior angles. This striking migration of the scapulae is the evident result of an almost complete absence of both trapezii. Exceedingly little musculature is to be felt between the vertebral column and the vertebral borders of the scapulae even when movements are made that ordinarily contract this musculature. Despite the defective trapezii, both arms can be strongly elevated above the head and retained there against considerable resistance.

Nevertheless, both scapulae are at the normal elevation. The left is of normal size (21.5 cm. by 14 cm.) and shape and the index is approximately normal (65.11). On the other hand, the right scapula is broader than normal (21.5 cm. long by 17.75 cm. wide) and, accordingly, presents an abnormally high index (82.55). In addition, its superior angle is very narrow and sharp, making a pointed spur that is readily felt through the supraspinatus muscle.

The right upper arm of this individual is much smaller than the left (circumference of the left, 27 cm.; of the right, 19 cm.), owing to extensive defects in

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6. In the usual types of scoliosis, the scapula on the side of the convexity is of course generally higher. It is of interest that the scapula on the side of the concavity of a scoliosis is generally higher in those instances in which unilateral Sprengel's deformity is combined with scoliosis. Several of the cases here presented will illustrate this.

7. It is regrettable that the photographs of this case were so imperfect that they were useless for reproduction. This individual and his children were examined under disadvantageous circumstances.

both the biceps and triceps. Nevertheless, the functions of the arm can be carried on in the usual manner, the power both of flexion and extension of the forearm being excellent.[8]

IV. CONGENITAL ELEVATION OF BOTH SCAPULAE

CASE 4.—Mrs. L., 45 years old, undersized and very obese. She had had a post-partum infection with a secondary intraabdominal abscess following the birth of her second child (Edward L., Case 17) and metrorrhagia since that time. She has never been able to abduct the right arm beyond the horizontal without rotating it inwards; the movements of the left arm have always been unrestricted.

The general physical examination is negative with the exception of a fibrid uterus, and the evidences of a poor peripheral circulation. The face of this individual is markedly asymmetrical, the left side being lower and more flat than the right (Fig. 6). There is a slight right-sided torticollis that can be overcome by a voluntary effort.

Both scapulae are displaced upwards, the left more than the right. On the other hand, although both are abnormally distant from the median line, the right is more so than the left. The details follow:

The *right scapula* (20 cm. long, 15.5 cm. wide; index, 77.5) lies between the levels of the seventh cervical and the sixth dorsal vertebrae. The superior angle is prolonged into a remarkably thin, sharp spur that can be felt in the supraclavicular fossa about 3 cm. above the clavicle. Although partly cartilaginous, this prolongation is more ossified than in Case 1 (Fig. 7). The supraspinata, 8 cm. in length, and the infraspinata, 12 cm. long, make a subscapular angle only slightly less than normal (120 degrees). The measurements above given, however, indicate the very pronounced disproportion in size between the supra- and infraspinata. The marked forward tilt of the scapula (to an angle of 30 degrees from the vertical) does not result in any visible deformity in this stout individual. It has already been stated that the right scapula is further from the median line than the left. At the superior angle it is 13 cm., at the scapular spine 14 cm., and at the lower angle 15 cm. from that line.[9] Concerning the musculature of the right scapula, a slight deficiency in the serratus magnus and the lower part of the trapezius could be determined; the levator anguli scapulae seems normal. The right arm can be abducted to an angle of 90 degrees. Full abduction is possible only by extreme inward rotation com-

8. The absence of any distinct loss of function despite the defects in both trapezii, the right triceps, and the right biceps of this individual illustrates what appears to me to be a fairly general law about congenital muscle defects. I have seen a number of instances of muscle deficiencies in various parts of the body in which the function of the parts remained approximately normal and I could only conclude that the function must have been taken up by other muscle-groups. To illustrate, a striking instance which I saw through the courtesy of Prof. M. Lewandowsky of Berlin may be mentioned. The patient had complete absence of the right pectoralis major and minor muscles. The arm could be used in a normal manner, with the full power of adduction of the limb. Other examples will be found in a number of the cases of Sprengel's deformity described in this paper; it was, at first, surprising to me to find that so little disability resulted from extensive muscle defects in several of these individuals. The question of the existence of a general law such as I have indicated is, perhaps, worthy of investigation, for I cannot find that it has been demonstrated.

9. Owing to the dorsal scoliosis, the spinous processes could not be employed as a guide to the median line in this case.

bined with a swinging motion: the patient cannot slowly abduct her arm above the horizontal. In abduction the scapula glides far from the median line.

The deformity of the *left scapula* (19 cm. long, 15 cm. wide; index 78.41) resembles that of the right. It is, however, 2 cm. higher, extending 5 cm. above the clavicle. The left shoulder is correspondingly higher than the right (Fig. 6). Measured on the spinal column, the left scapula extends from the sixth cervical to the sixth dorsal vertebra. Its longitudinal axis makes an angle of 20 degrees with the perpendicular. The superior angle is prolonged in a spur that is even

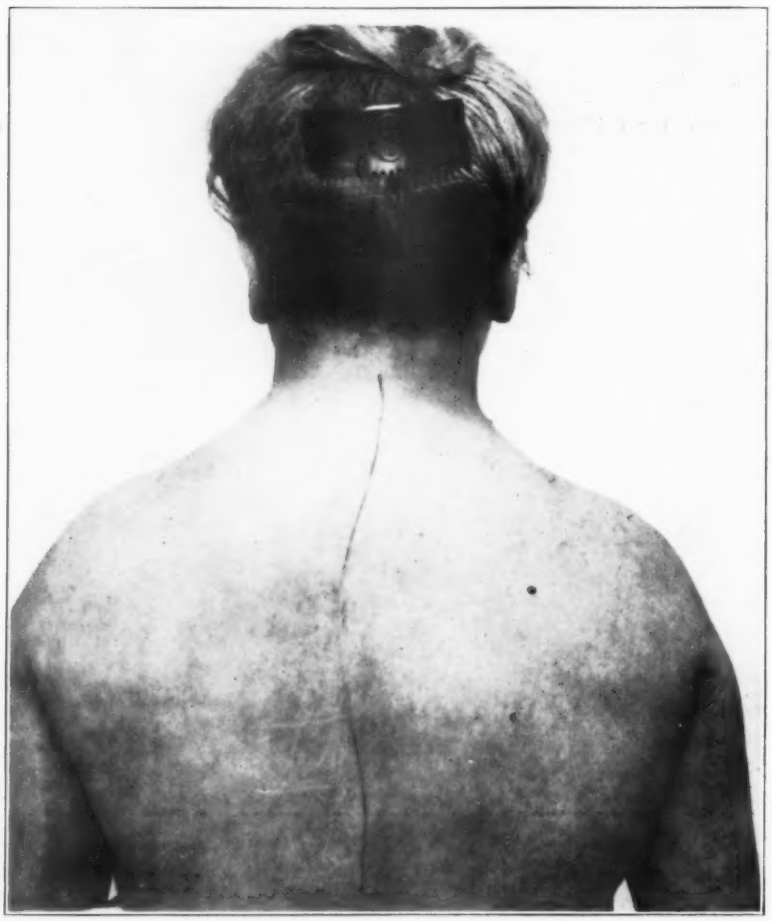

Fig. 5.—(Case 4) Mrs. L., rear view. The line indicates the position of the spinous processes.

more prominent than the one on the right side (Fig. 7). At the tip this spur presents a peculiar hook that points directly backwards: this is faintly visible in the radiograph. The supraspinata (7 cm. long) and the infraspinata (12 cm. long) make a subscapular angle of about 115 degrees. Although abnormally distant from the median line, the left scapula is nearer that line than the right: the measurements from the mid-line are 12.5 cm. at the superior angle, 12 cm. at the spine of the scapula, 12.5 cm. at the inferior angle. The musculature and movements of the left arm are apparently normal.

Fig. 6.—(Case 4) Mrs. L., front view.

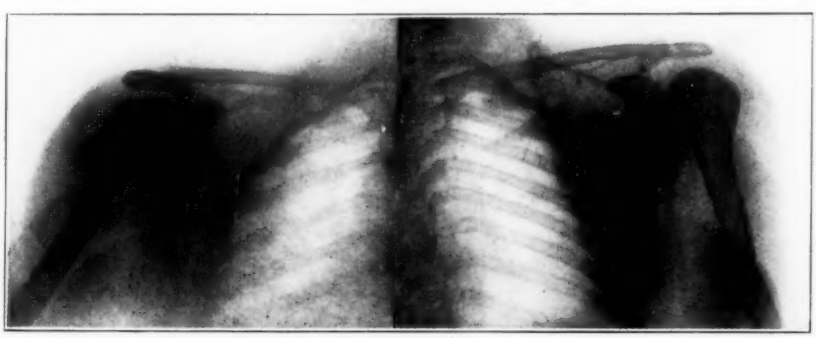

Fig. 7.—(Case 4) Mrs. L. Simultaneous radiographs of right and left scapulae, taken on separate plates in order to minimize inaccuracies.

The most striking remaining deformity is a sharp left lateral scoliosis of the mid-dorsal spine. This cannot be well seen in the photograph of the patient's spine, nor is it well seen in the radiographs. It was more clearly visible in the radiographs of the spinal column taken to determine if anomalies in the number, distribution, etc., of the vertebrae were present. No such anomalies were found. It should be noted that the intercostal spaces are quite even despite the scoliosis, a fact that indicates the congenital nature of the spinal curvature. Finally, it should be mentioned that both clavicles are short, thick and straight; the left one (14 cm. long) is 1.5 cm. shorter than the right.

To summarize the findings in this case:

	Right Scapula	Left Scapula
Measurements	20 by 15.5 cm.	19 by 17 cm.
Index	77.5	78.41
Superior scapular angle	3 cm. above clavicle; very prolonged and sharp.	5 cm. above clavicle. Very prolonged, sharp; hook-like process at tip.
Supraspinata	8 cm.	7 cm.
Infraspinata	12 cm.	12 cm.
Subscapular angle	120° (approximately)	115° (approximately)
Vertebral border of scapula from mid-line at:		
Superior angle	13 cm.	12.5 cm.
Spine	14 cm.	12 cm.
Inferior angle	15 cm.	12.5 cm.
Rotation on frontal axis	30°	20°
Serratus magnus	Deficiency (Not marked)	Normal
Trapezius	Deficiency (Not marked)	Normal
Abduction of arms	To 90°	Normal

The other features of interest are: Facial asymmetry. Dorsal scoliosis combined with normal intercostal spaces. Short, incurved clavicles, the left shorter than the right.

V. LEFT-SIDED SPRENGEL'S DEFORMITY

CASE 5.—Ruth H., 3 years old, obese. Has had several attacks of generalized epilepsy of non-focal type, beginning at the age of 2. Excepting a slight alternating strabismus, the general examination is negative. The scapulae are normal in shape and size; the left, however, is 2 cm. higher than its fellow. This elevation is the same when measured at the superior and the inferior angles and at the scapular spines. The left scapula lies between the levels of the seventh cervical and the sixth dorsal vertebrae; the right, between the first and the seventh dorsal vertebrae. Both scapulae are equidistant from the median line and the movements of both upper extremities are normal.[10]

VI. SLIGHTLY "WINGED" SCAPULAE

CASE 6.—Jerome H., 8 years old. Ribs, radius, ulna, skull, etc., show evidence of previous rickets. The inferior angles of both scapulae are distinctly more prominent than normal, but, in the absence of any other abnormalities in the scapulae or in their functions, no great significance can be placed on this somewhat "winged" appearance of the shoulder-blades. There were no discoverable defects in the trapezii or serrati, but the musculature could not be tested by electrical stimulation in this case.

10. Photographs and roentgenograms of this and the other members of this branch of the family could not, unfortunately, be obtained.

VII. LEFT-SIDED SPRENGEL'S DEFORMITY

CASE 7.—Pauline H., aged 10 years. No illnesses other than the ordinary ones of infancy. The elevation of the left shoulder has been observed since the child was 1 year old; it has not apparently increased in the succeeding years.

There is a slight left lateral curvature of the spine in the mid-dorsal region. The left scapula is 3 cm. higher than the right and the shoulder is correspondingly elevated. The right scapula is normal in size, shape, musculature and position. Its vertebral border lies 3.5 cm. from the median line and extends from the level of the second dorsal vertebra to that of the eighth. The left scapula is normal in shape, but is smaller than its fellow. It lies between the levels of the last cervical and the sixth dorsal vertebrae and is further from the median line than the right scapula (the distance at its superior angle being 2.5 cm.; at the scapular spine, 4 cm.; and at the inferior angle, 7 cm.) owing to a rotation on its frontal axis. The rotation does not appear so marked because the vertebral column is also bent to the left in the dorsal region. The inferior angle of the left scapula is much more prominent than that of the right, giving the left shoulder a somewhat "winged" appearance. Electrical stimulation shows that the cause of the latter is a partial defect in the serratus magnus and in the lower portion of the trapezius. There is some weakness in the power of forcing the left arm forwards; and the inferior angle of the scapula cannot be drawn back to the vertebral column (a hiatus of 4.5 cm. remaining). The absence of musculature is also noticeable on palpation. The grosser movements of the left shoulder-girdle are quite normal, however.

VIII. THORAX OF CYLINDRICAL CONTOUR

CASE 8.—Sidonia H., 12 years old, undersized and underweight. Her thorax corresponds very remarkably with that of her father (Case 1). It is quite cylindrical, the circumferences at different levels being all practically the same. The ribs slope sharply downwards, but not as markedly as in the parent. Her shoulders are very sloping, but both scapulae are normal in size, shape, position and function.

IX. CONGENITAL ELEVATION OF THE SCAPULAE OF SLIGHT GRADE

CASE 9.—Gertrude H., 13 years old, poorly nourished. Her thorax is long and is flattened anteroposteriorly. The spinal column is normal. Except for their position, the scapulae do not present any abnormality in size, shape or function.

Both scapulae are elevated above the normal position, the left more than the right. The latter lies between the levels of the first and the seventh dorsal vertebrae; the former, between the seventh cervical and sixth dorsal vertebrae. Both shoulders are correspondingly elevated. Were these findings noted in an individual not a member of a family suffering from Sprengel's deformity, the classification of the case as one of bilateral congenital elevation of the scapulae would not be justifiable.[11]

X. KYPHOSIS. DEFORMITY OF THORAX

CASE 10.—Israel H., aged 6 years. History of rachitis in early childhood; evidences of the disease in the long bones, skull, etc. The chest is markedly deformed, the apparent result of a forward bulging and angulation of the sternum that seems rachitic in nature. The spinal column presents a rounded dorsal kyphosis that is very marked; there is a slight compensatory lumbar lordosis. Excepting slight limitaton in flexion and extension, the movements of the spine are normal. The scapulae are normal in size, shape and position.

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11. It is interesting to note that Sprengel believed that the scapulae were invariably normal in the deformity to which his name has very generally been given. Subsequent observers, however, demonstrated that the scapulae are usually (though not necessarily) distorted in Sprengel's deformity.

XI. LEFT-SIDED SPRENGEL'S DEFORMITY, DEFECTIVE MUSCULATURE ON THE RIGHT SIDE

CASE 11.—Edward H., 10 years old, presents an elevation of the left shoulder that has been observed since earliest infancy. His physical condition is good, and, with the exception of numerous moles scattered over the body, the general examination is negative. The spinal column is normal. Both scapulae are

Fig. 8.—(Case 11) Edward H., rear view.

abnormally distant from the spinal column, the left more so than the right (Fig. 8). The left scapula is 5 cm. from the mid-line at the superior angle, 6.5 cm. at the scapular spine, and 8 cm. at the inferior angle. These measurements indicate the rotation that exists on the frontal axis. The inferior angles of the scapulae are more prominent than normal, and this is more pronounced

on the right side. By employing the usual tests, a partial deficiency in the serratus magnus and in the lower part of the trapezius can be determined on *both* sides. The grosser movements of the shoulder-girdles are normal, however. The left scapula is 2.5 cm. higher than the right.[12] It lies between the last cervical and the seventh dorsal vertebrae, and is somewhat longer than the right scapula. The left supraspinata, however, is 2 cm. shorter than the right, and is bent forward abnormally, the subscapular angle being about 115 degrees. The left superior angle is readily palpated; it lies 1.5 cm. above the clavicle and is much broader and more rounded than the normal.[13] The indices of both scapulae are normal. The single series of radiographs that could be taken in this case did not show the abnormality of the left scapula satisfactorily.

XII. LEFT-SIDED SPRENGEL'S DEFORMITY

CASE 12.—Clara H., 1½ years old; birth normal. The mother looked for a difference between the shoulder-blades soon after the child was born, and noticed that the left was higher; she believes that the elevation increased in the succeeding months. It has also been noticed that the child cannot bring her left elbow to her side.

Fig. 9.—(Case 12) Clara H., rear view.

Owing to its extreme restlessness, careful examination of the child was impossible and this report is accordingly very incomplete. It was evident, however, that both scapulae (and especially the left) were abnormally distant from the median line and that the left scapula was at least 2 cm. higher than the right (Fig. 9). The superior angle of the left scapula could be distinctly felt about 2.5 cm. above the upper border of the clavicle. Both scapulae appeared normal in size and shape.

The left arm cannot be completely adducted to the side of the body, an angle of about 15 degrees between the axis of the arm and the left margin of the chest remaining. The arm can be carried along the front of the chest, however. There is no evidence of disease in or about the left shoulder-girdle and it is therefore thought that the inability to adduct the arm depends on extensive defects not only in the trapezius, but also in the muscles that arise from the scapula and are inserted into the humerus.

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12. The elevation of the scapula is not so evident in the photograph because the shoulder is not correspondingly elevated. This peculiarity is not infrequently observed in Sprengel's deformity.

13. This condition of the superior angle of the scapula is normal in the lower vertebrates.

XIII. RIGHT-SIDED SPRENGEL'S DEFORMITY

CASE 13.—Lillian H., 8 years old; elevation of the right shoulder-blade observed since infancy, but no other abnormal conditions noted. The right scapula is 2.5 cm. higher than the left; it lies between the levels of the last cervical and the sixth dorsal vertebrae. The superior angle, of normal shape, lies immediately above the upper border of the clavicle. The scapular index and the relations between the supra- and infraspinata are normal. The right scapula is somewhat further from the median line than the left (from median line to right superior angle, 7 cm.; to left superior angle, 6 cm.; to right inferior angle, 5 cm.; to left inferior angle, 3.75 cm.). Both inferior scapular angles are much more prominent than normal (Fig. 10); this is evidently due to defects in both trapezius muscles. The movements of the scapulae are quite normal, however. Unfortunately, there was no opportunity to make electrical tests of the musculature in this case.

The physical examination disclosed no abnormalities other than those of the scapulae.

Fig. 10.—(Case 13) Lillian H., rear view.

Fig. 11.—(Case 14) Edward H., rear view.

XIV. RIGHT-SIDED SPRENGEL'S DEFORMITY

CASE 14.—Edward H., 12 years old; curvature of the spine and elevation of the right shoulder-blade noticed since earliest infancy. The general physical examination was negative. The spinal column presents a moderate scoliosis in the lower dorsal region and a marked lordosis in the dorsolumbar zone. Motions of the spine are free in all directions.

The right scapula and shoulder are congenitally elevated, but the remarkable feature of this case is the difference in size between the two infraspinata. The right scapula is 15.75 cm. long and 12.75 cm. wide (index 80.08), and lies between the levels of the seventh cervical and the sixth dorsal vertebrae. Its musculature appears normal. Nevertheless, it is abnormally distant from the median line — 6.5 cm. at the superior angle and 8 cm. at the inferior angle. The right scapula is 2 cm. higher than the left at the superior angle and at the scapular spine, but it is 4.5 cm. higher at the inferior angle. This is due to a defect in the right infraspinata. The latter, trowel-like in shape, is considerably shorter and narrower than the normal. It is 10 cm. in length, whereas the left

infraspinata measures 12.75 cm. (Fig. 11). The right supraspinata is of normal shape; the superior angle lies about 2 cm. above the upper border of the clavicle. The angle between the supra- and infraspinata is approximately normal. The summary of the examination of this individual is as follows:

	Right Scapula	Left Scapula
Measurements	15.75 cm. by 12.75 cm.	18 cm. by 12.75 cm.
Index	80.09	70.83
Superior scapular angle	2 cm. above clavicle; normal shape.	Normal position and shape.
Supraspinata	5.75 cm.	5.25 cm.
Infraspinata	10 cm.; trowel-shaped.	12.75 cm.; normal shape.
Vertebral border of scapula from mid-line at:		
Superior angle	6.5 cm.	6 cm.
Inferior angle	8 cm.	6 cm.
Subscapular angle	Approximately normal.	Approximately normal
Musculature and movements	Normal	Normal.

The only additional features are the marked lordosis and the moderate scoliosis to the right.

XV. NORMAL INDIVIDUAL

Case 15.—Gertrude H., 14 years old, of good physique. Examination is entirely negative. She is the only member of the whole family in whom an abnormality of one form or other is not to be found.

XVI. ANOMALY OF THE LEFT MAMMA

Case 16.—Morris L., aged 14 years; very muscular. The spine, thorax and scapulae are normal. The left breast is somewhat larger than the right. Around its nipple is an areola much greater in area and far more deeply pigmented than the normal, the contrast between the two breasts being very striking. The tissues in and about the left nipple are firm and fibrous. The right testicle of this individual is much lower than the left. No other abnormalities are to be found.

XVII. BILATERAL CONGENITAL ELEVATION OF THE SCAPULAE

Case 17.—Edward L., 15 years old. He has had epileptiform seizures for five years, not well controlled by the bromid therapy employed. This patient was the first of the family to appear in the Surgical Clinic of Mount Sinai Hospital. He came because he was unable fully to abduct the right arm. Examination revealed the presence of bilateral Sprengel's deformity. I then considered it possible that other members of his family might also present congenital malformations and, in the examination of them, determined the findings reported in this paper.

As seen in the photograph (Fig. 12), the facial asymmetry of this patient resembles that of his mother (Case 4) and of his uncle (Case 1). The heart occupies an abnormally central position and is somewhat smaller than normal.[14] The hard palate is narrow and very highly arched. There is a fibrous tumor of the second toe of the left foot, said to have been present at birth. It is not con-

14. The examination of the heart was made by Dr. B. S. Oppenheimer of New York, to whom I am indebted for this report.

nected with the bone, the Roentgen-ray picture of the foot being normal. The pelvis of this patient shows a congenital narrowing, to be seen in the radiograph (Fig. 14). The general examination reveals no other malformations. The detailed examination of the scapulae follows:

The *right scapula* is 2.5 cm. higher than the left and extends from the level of the seventh cervical to that of the sixth dorsal vertebra. It is 17 cm. long and 15 cm. wide (index 88.24), and shows an abnormal rotation both on the sagittal and on the frontal axis; the vertebral border is 14.5 cm. from the median line at the superior angle; 11 cm. at the scapular spine; 10 cm. at the inferior angle. The scapula is tilted forward to an angle of 45 degrees with the vertical. The supraspinata (5 cm. in length) is bent forward on the infraspinata (12 cm.

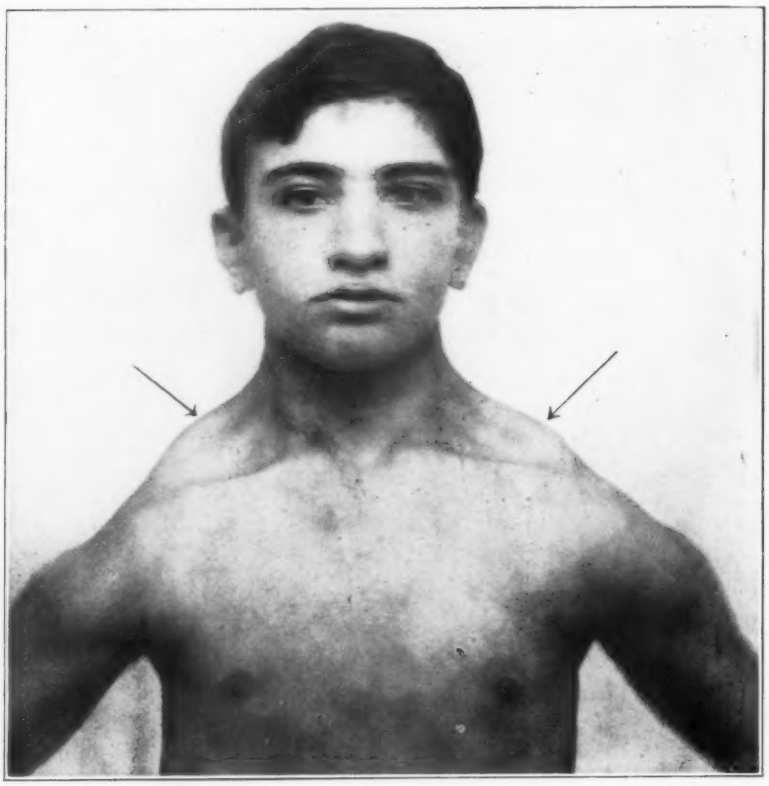

Fig. 12.—(Case 17) Edward L. Arrows point to the superior scapular angles. Right arm is abducted to the full degree.

long), making a subscapular angle of 110 degrees. The superior angle, rising 5 cm. above the clavicle, is very prominent (Fig. 12). It is surrounded by a rounded, cartilaginous cap. The bony portion, however, is also abnormally and peculiarly rounded (Fig. 13). There is a very pronounced deficiency in both the trapezius and serratus magnus muscles and, very probably, in the rhomboid muscles also. As a result, the right arm cannot be normally abducted beyond an angle of 45 degrees; further abduction can be accomplished only by turning the body away from the right arm and then swinging the arm upwards. The full degree of abduction of the *right* arm is shown in the photograph (Fig. 12).

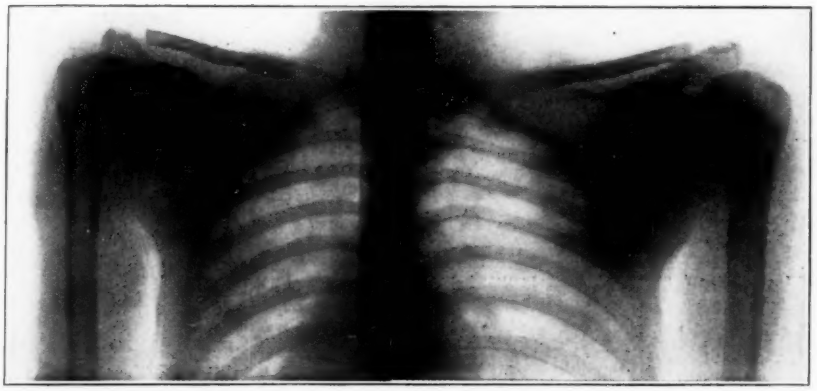

Fig. 13.—(Case 17) Edward L. Simultaneous radiographs of right and left scapulae, taken on separate plates in order to minimize inaccuracies.

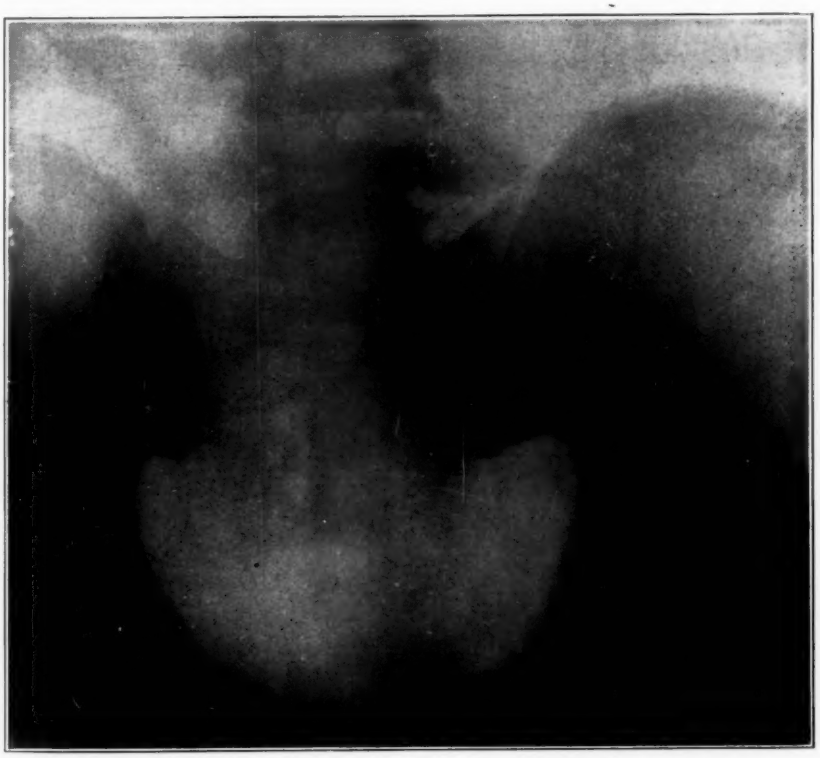

Fig. 14.—(Case 17) Edward L. Congenitally narrow pelvis.

The scapula slides far from the median line as soon as abduction begins; it is almost completely *inverted* when the arm is brought up beside the head.

The *left scapula* does not present as extreme a picture as the right. It is 19 cm. long, 14 cm. wide (index 73.16), and lies between the levels of the seventh cervical and seventh dorsal vertebrae. The superior angle, palpable 2 cm. above the upper border of the clavicle, is slightly rounded and more extensively cartilaginous than that of the right shoulder-blade. The supraspinata (measuring 6 cm.) is bent forward on the infraspinata (13 cm. long), making a subscapular angle of 110 degrees. The left scapula is only slightly tilted on the frontal axis (10 degrees). It is abnormally distant from the median line (12.5 cm. at the superior angle, 10.5 cm. at the scapular spine, 9.5 cm. at the inferior angle), but is not as far from the median line as the right scapula, nor does it show the *combined* sagittal and frontal rotation there observed. The trapezius and serratus magnus are extensively deficient, but this, too, is not as pronounced as on the right side. The migration of the left scapula in even moderate abduction of the arm is seen in the photograph (the arrows pointing to the superior angles of the scapulae), yet full abduction, adduction, forward elevation, etc., are possible. The weakness of the lower part of the trapezius and of the serratus magnus is very distinct, however.

The remaining abnormalities of the thorax are: The right pectoralis major is distinctly smaller than the left. Both clavicles are uncurved and are thickened at their distal ends. The ribs are almost horizontal in position, even the lowermost showing barely any downward slant.

The summary of the abnormalities found are as follows:

	Right Scapula	Left Scapula
Measurements	17 cm. by 15 cm.	19 cm. by 14 cm.
Index	88.24	73.16
Superior scapular angle	5 cm. above clavicle; prominent; rounded; cartilaginous.	2 cm. above clavicle; not so prominent; slightly rounded; more cartilaginous.
Supraspinata	5 cm. long.	6 cm. long.
Infraspinata	12 cm. long.	13 cm. long.
Vertebral border from mid-line at:		
Superior angle	14.5 cm.	12.5 cm.
Spine of scapula	11 cm.	10.5 cm.
Inferior angle	10 cm.	9.5 cm.
Rotation on frontal axis	45°	10°
Serratus magnus	Marked deficiency.	Deficiency not so marked.
Trapezius	Marked deficiency in lower half.	Deficiency not so marked.
Abduction of Arm	To 45°	Complete
Pectoralis major	Small.	Normal size.
Clavicle	Uncurved; distal end thickened.	Uncurved; distal end thickened.
Ribs	Almost horizontal.	Almost horizontal

The other abnormalities are: Facial asymmetry; highly arched palate; small heart situated more mesially than the normal; narrow pelvis; tumor of toe of congenital origin.

XVIII. DEFORMITY OF THE SCAPULAE AND OF THE THORAX. NO SPRENGEL'S DEFORMITY

CASE 18.—Harry L., 17 years old, very muscular individual. Although right-handed, the left half of his thorax is evidently wider than the right (compare Roentgen-ray pictures, Fig. 15). At the the level of the nipples the arc of the left half of the thorax measures 45.5 cm.; of the right half 40 cm. The distance from the sternal notch to the tip of the left shoulder is 20.5 cm.; to the tip of the right shoulder, 17.5 cm. The ribs and intercostal spaces are normal. The heart is normal in position.

Both scapulae are on the same level, but are on an elevation slightly higher than the normal. Each presents an abnormally high index — the right (18.75 cm. long and 13.25 cm. wide) 70; the left (18.25 cm. by 12.50 cm.), 69.24. The measurements indicate the slight difference in size. The superior angles of both bones are abnormally rounded, the right more so than the left. Both are more than normally distant from the median line, especially the right. The latter

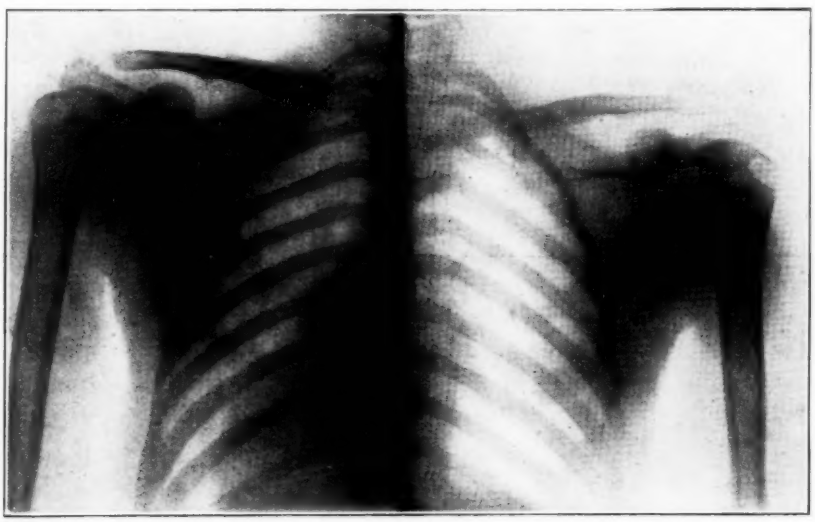

Fig. 15.—(Case 18) Harry L. Radiographs of right and left scapulae.

is 13 cm. from the median line at the superior angle; 14.5 cm. at the inferior angle; the former 10 cm. from the mid-line at the superior angle and 11 cm. at the inferior angle. A peculiar isolated area of bone is to be seen in the radiogram, in the inferior angle of the left scapula.[15] A slight deficiency in the lower portion of the left trapezius muscle can be definitely determined; the musculature of both scapulae is otherwise normal. The bones show no abnormal rotation on the frontal axis and no distortion of the subscapular angles.

To summarize the findings: Left half of thorax much wider than right. Abnormally high indices of both scapulae. Rounding of the superior angles. Abnormal distance of both scapulae from the median line (especially the right). Partial deficiency of left trapezius. Peculiarity of inferior angle of left scapula (seen in Roentgen-ray picture).

15. This peculiar appearance was also seen in another Roentgen-ray picture taken to establish the finding.

XIX. LEFT-SIDED SPRENGEL'S DEFORMITY OF SLIGHT GRADE

CASE 19.—Esther L., 20 years old, of good physique. The right scapula is normal in size, shape and position. The left shoulder is slightly higher than the right. The *left scapula* is 3 cm. higher, and 1.5 cm. nearer the median line than the right. It lies between the levels of the first and seventh dorsal vertebrae. The superior angle, of normal configuration, is palpable 1.5 cm. above the upper border of the clavicle. The index of the left scapula, the musculature, and the relations between the supra- and infraspinata are all normal. The vertebral column is also normal.

DISCUSSION

As indicated in the introduction to this paper, it was not primarily with the object of adding a number of cases of unilateral and bilateral congenital elevation of the shoulder that the above cases have been presented. Although this material offers a number of unusual features, it does not in itself add much to our knowledge of the affection in question. Indeed, a study of the subject will disclose a number of variations of the picture of Sprengel's deformity which my cases do not illustrate.[16] On the other hand, however, the presence of several instances of congenital elevation of the scapula in one family throws a new light on the etiology of this affection. The present knowledge of the etiology of Sprengel's deformity should be briefly reviewed in order to prove the truth of this assertion.

A number of theories have been advanced to explain the causation of the deformity. Sprengel believed that the deformity depended on a subnormal amount of amniotic fluid. When an arm of the fetus was accidentally forced backwards it could not float free, became permanently fixed in that position, and the high scapula resulted. According to Kirmisson, the elevation of the scapula is secondary to malformation of the bone. The latter, together with an arrest of development, is due to amniotic bands (Schlange), to a periostitis or an osteoma of the supraspinata (Eulenberg), etc. The more recent theories base the high position of the scapula on failure of descent. Thus Rager believes that disease of the superior part of the scapula, or an anomalous position in which it lies, so changes its shape that the bone, being incapable of descent, retains its fetal position.[17] According to Kausch, the lower part of the trapezius muscle is the principal factor in retaining the scapula in its normal position; he believes that a primary defect of the trapezius exists in all cases of Sprengel's deformity, and that the elevation of the scapula is secondary to the muscular defect. In

16. A complete summary of the varied manifestations of Sprengel's deformity is to be found in a paper by Horwitz, Am. Jour. Orthop. Surg., November, 1908. I have drawn on this paper for many of the facts that have been quoted.

17. The scapula of the normal fetus occupies a cervical position and only descends to its thoracic position during the last months of fetal life and the early months of infancy.

Horwitz' analytical survey of the literature of the subject the most plausible aspects of each theory are combined. His combined theory for the etiology is:

1. That it is failure of descent caused by

(*a*) Too great intra-uterine pressure, due either to an increased or diminished amount of amniotic fluid.

(*b*) Abnormal articulations of the scapula with the vertebral column.

(*c*) A defective musculature, unable to draw the scapula caudally, where neither (*a*) nor (*b*) exists.

(*d*) A normal musculature, unable to draw the scapula caudally where either (*a*) or (*b*) exists.

2. That the changes in the shape and size of the scapula are an arrest of development due to improper or defective muscular tension.

A complete analysis of these theories is foreign to the purposes of this paper. It is enough to state that they are based chiefly on the anatomical peculiarities that have been found, but they do not suggest any underlying cause for these abnormalities.

The presence of several instances of congenital elevation of the scapula in one family clearly demonstrates, for that family at any rate, that the affection is closely allied to various other (degenerative) inherited or familial affections. It also demonstrates that the unilateral and the bilateral congenital high shoulder are varieties of one affection, although the latter type is much more rarely seen than the former.

In the family which I have described, two features were regularly present in those members who were pronounced examples of the deformity: First, malformation of the scapula, and, second, defect of the trapezius and serratus magnus muscles. The questions that arise are, therefore: 1. Which is the primary lesion, that of the bone or that of the muscle? 2. Are the lesions of bone and muscle independent, each being a part of a more general, underlying fault of development?

Although Sprengel believed that the scapula was unaltered in shape in the deformity he described, many subsequent observers have noted alterations in the size, shape and contour of the bone. According to Horwitz, 39 per cent. of all recorded cases have shown an abnormality in the relations between the two diameters of the scapula; he believes that the change "would probably have been found in all cases had careful observation been made. Curving of the superior border and the forward bending of the supraspinata were seen in 18.5 per cent. of the cases. A bony, fibrous or cartilaginous attachment of the scapula to the vertebral column" was found in 25 per cent. of the cases. Although a trapezial defect may account for the high position of the scapula, it is inconceivable that that will explain the deformities of the bone that have been encountered as frequently as above indicated. Besides,

a weakness of or defect in the trapezius was observed in only 33.8 per cent. of all reported cases.[18] It is equally impossible to ascribe the commonly met lesion in the trapezius to the deformity of the scapula.

It is therefore far more logical to believe that these lesions are not interdependent, but that each is part of a more general fault of development. This viewpoint is strongly supported by the familial type of Sprengel's deformity that has been described.

What is the nature of this underlying fault of development? According to Minot and other embryologists the scapula (and its musculature as well) develops in a sheet that is continuous with the vertebral column. Theoretically, then, some defect in the sheet should lead to an anomalous condition of the musculature connected with the scapula (especially the important trapezius and serratus magnus muscles). An aberrant center of ossification developing in the sheet would result in abnormal articulations of the scapula with the spine. The bending of the supraspinata would be explained by the moulding of that part of the bone lying in the cervical musculature.

Such theoretical reasoning, however, will not account satisfactorily for (1) the alterations in the diameters of the scapula; (2) the other deformities so often encountered in instances of Sprengel's deformity (facial asymmetry, scoliosis and other spinal deformities, rib defects, etc.). Concerning the first it should be pointed out here that the normal fetal scapula presents a greater length in the transverse than in the vertical diameter. Without entering further into this interesting aspect of evolution, it may be stated that an arrest of fetal development of the scapula would account for its diameters as found in many cases of Sprengel's deformity. However, the scapula in these cases does not generally present the picture of arrested development, but rather that of distorted development. This scapula, distorted for the human being, is on the contrary normal in shape for the anthropoid ape,[19] and in some vertebrates even lower in the scale. Therefore, when several such scapulae are found in members of one family the inevitable conclusion is that Sprengel's deformity is, under these circumstances at any rate, a reversion to a lower type. The deformities, other than those met with in the scapula, must be considered (in the family described) as incidental faults of development in a family subject to atavism.

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18. Kausch believes that an involvement of the trapezius will be found in every case of Sprengel's deformity if the examination is carefully made. Nevertheless this did not hold true in several I have described and in some of the cases described by others.

19. According to Broca, the scapular index of the European is 65.91; of the chimpanzee, 68.52; the gorilla, 70.38; the gibbon 96.97, etc. (Quoted from Horwitz.)

It would be a far cry to pronounce all instances of Sprengel's deformity reversion to a lower type, on the basis of atavism found in one family subject to the deformity. I firmly believe, however, that other instances of the deformity (or of other evidences of degeneracy or of reversion) would not infrequently be encountered if the *families* of individuals the subjects of Sprengel's deformity were thoroughly examined.[20] The reports of such examinations are surely indicated, for they will greatly aid in casting additional light on the etiology of congenital elevation of the scapula.

RÉSUMÉ

The features of interest in the individual cases cannot be summed up briefly and must therefore be sought in the body of the paper. As a result of the study of these cases it is concluded that:

1. Congenital elevation of the scapula occurs in a familial form.

2. Unilateral and bilateral congenital elevation of the scapula are varieties of one affection.

3. The deformity of the scapula and the defects in the trapezius (as well as other coexisting anomalies) are parts of a more general fault in development, i. e.,

4. Sprengel's deformity is a manifestation of atavism when it prevails in a familial form. It is probably a manifestation of atavism when it occurs in isolated instances.

5. If sought for, more instances of congenital elevation of the scapula of familial type will in all probability be found. Important additional knowledge of the etiology of the affection will thereby be acquired.

I wish to express my thanks to Dr. W. M. Brickner, formerly Chief of the Surgical Department of the Mt. Sinai Hospital Dispensary, for his courtesy in permitting me to report the first case of Sprengel's deformity that appeared in his clinic; and indicate my indebtedness to Dr. L. Jaches, radiographer to Mt. Sinai Hospital, without whose cooperation in the radiographic studies this work could not have been accomplished.

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20. It is, for example, very regrettable that Sick (Deutsch. Ztschr. f. Chir., 1902, No. 67, p. 566) did not examine the other members of a family, one of whom came under his observation with the typical picture of bilateral Sprengel's deformity. In his report Sick states that "the mother (of the child affected with the deformity) states that she has had several children, all normally born, and that the patient was the only one who showed the shortening of the neck. Her husband and she are free from the deformity. But the husband's sister and several other members of his family have high shoulders very similar to those of the child." This is the only reference found in the literature in which the possibility of several instances of Sprengel's deformity occurring in one family is indicated.

RESPIRATORY INFECTIONS IN INFANTS' WARDS*

WALTER F. CHAPPELL, M.D., AND ALAN BROWN, M.D.
NEW YORK

The subject of ward infections in children's hospitals has long been one of discussion from the standpoint of contagious diseases, while the infectious nature of the respiratory affections has received little attention until recently. In the wards of the Babies' Hospital it is not measles, whooping-cough or scarlet fever that is feared, but rather the pneumococcus and streptococcus infections, both of which may be said to be omnipresent. In fact it seems evident that if we could prevent the respiratory infections, which begin as simple rhinitis or pharyngitis and frequently end in bronchopneumonia, we could save a much larger number of feeding cases (marasmus babies).

During the past two years we had 129 instances of acute nasopharyngeal infections in our feeding wards and not one single case of measles, whooping-cough or scarlet fever. During this same period we have lost from uncomplicated marasmus but twenty-nine out of 271 marasmus babies treated, while of the total deaths, ninety-seven in number, two-thirds were due to complications, chiefly respiratory.

INCREASE OF RESPIRATORY AFFECTIONS

A striking change seen in the last fifteen years in the Babies' Hospital has been the reduction in the proportion of intestinal diseases from 36 per cent. of the total admissions to 13 per cent., and an increase of severe acute respiratory infections from 8 per cent. to 17 per cent. This is only further evidence that the intestinal diseases are being prevented in the city at large, while the respiratory infections are not. Nothing in the hospital service is more discouraging than the development of acute nasopharyngitis, otitis, laryngitis, bronchitis and bronchopneumonia in infants admitted for other conditions. In fact, these infections are more serious than the common contagious diseases and are always more difficult to control, because the source of infection and manner of spreading are less obvious.

The following table shows the number, variety and mortality of the acute infections of the respiratory tract met with in 271 feeding cases, under treatment in two seasons:

* Submitted for publication March 5, 1914.

* From the Babies' Hospital, New York.

	Number	Mortality, Per Cent.
Bronchopneumonia	29	84
Otitis	63	60
Diphtheria	11	54
Bronchitis	4	75
Adenitis	4	0
Meningitis	3	100
Septicemia (only)	3	67
Nasopharyngitis (simple)	2	50

This table is not intended to convey the impression that the infants in every case died as a direct result of the complications, but rather that these infections played a large part in the final issue. In many instances they were the direct cause of death. Bronchopneumonia in these patients has an exceedingly high mortality, owing to their feeble resistance, while meningitis was always fatal.

The following table illustrates the relative frequency of the infection by months:

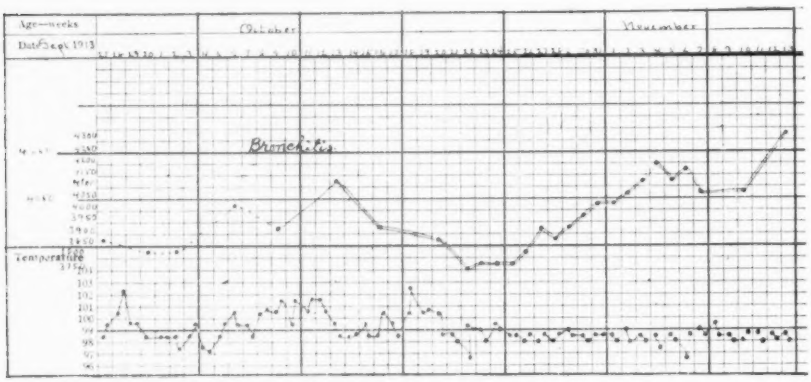
Month	Number of Infections	Percentage of Total No. of Infections
January	14	10.8
February	16	12.4
March	14	10.8
April	13	10.0
May	5	3.8
June	3	2.3
July	7	5.4
August	2	1.5
September	4	3.1
October	14	10.8
November	10	7.7
December	17	13.1

A somewhat similar chart compiled by L. F. Meyer in Berlin shows a corresponding frequency of ward infections during the fall and winter months in comparison with the warm season.

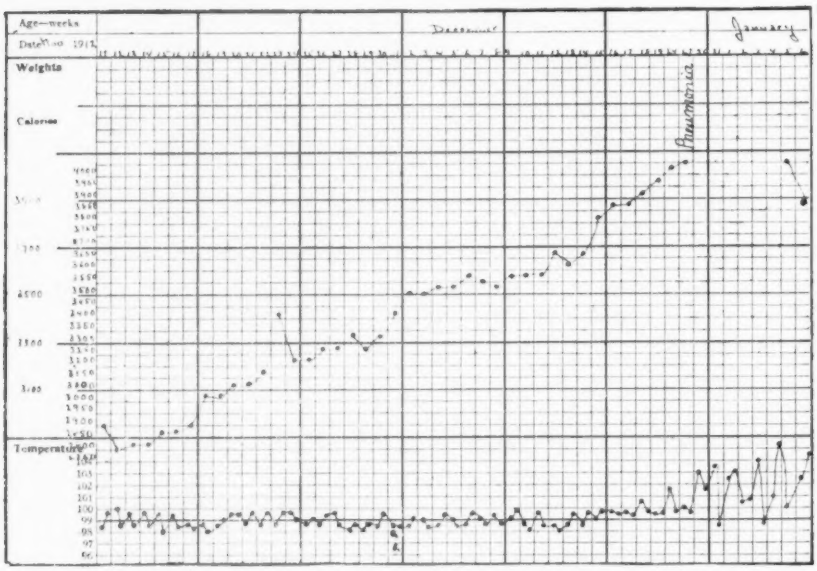
It has been a rather striking observation that the degree of susceptibility to infection varies directly with the length of residence in the institution. The average stay in the hospital for 271 feeding cases was 46.4 days, in those developing infections 57.3 days and in those developing more than one infection 65 days. It is worthy of note that one infection readily predisposes to the development of another. Out of 129 respiratory infections there were sixty-nine cases with only one infection, twenty-eight with two infections, and two cases with three infections. This last point has been further illustrated by Meyer, who showed that after the second week of hospital residence the infections gradually increased from 0.25 per cent. to 3.08 per cent. at the end of 100 days.

The following histories serve to illustrate what occurs throughout the year in working with these delicate infants:

CASE 1.—B. I., aged 7 months, weight 3,850 gm. For the four weeks following admission weight was practically stationary; during this period the infant had signs of a general bronchitis with a leukocytosis. Not until all signs had disappeared and the temperature had become normal did the infant begin to gain (Chart 1).



Weight and Temperature Chart of Case 1.



Weight and Temperature Chart of Case 2.

CASE 2.—M. S., aged 2½ months, weight 2,900 gm., gained 1,600 gm. in six weeks. Developed a bronchopneumonia and died in seven days (Chart 2).

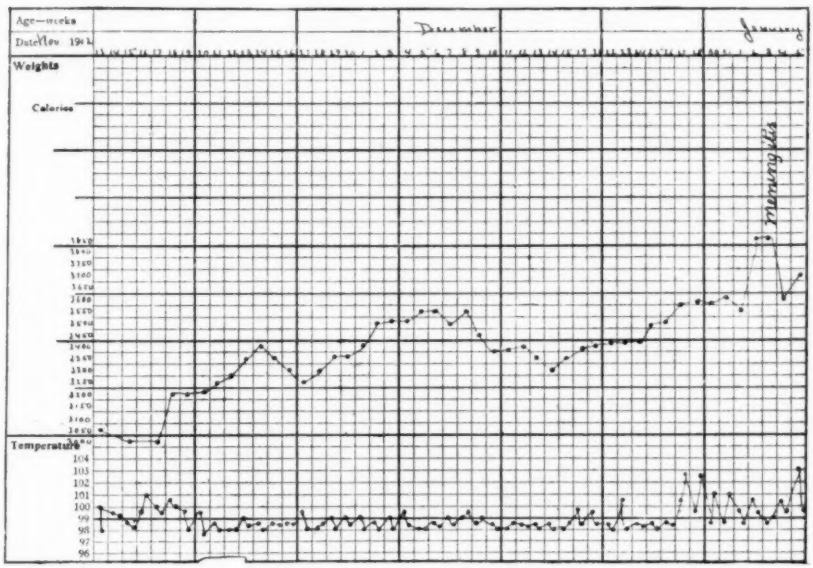
CASE 3.—S. W., aged 11 days, weight 3,025 gm., after having been in the hospital six weeks and gained 825 gm., developed streptococcus meningitis and died in two days (Chart 3).

CAE 4.—P. M., aged two weeks, weight 2,350 gm. On two successive occasions developed a blood infection due to the pneumococcus and staphylococcus

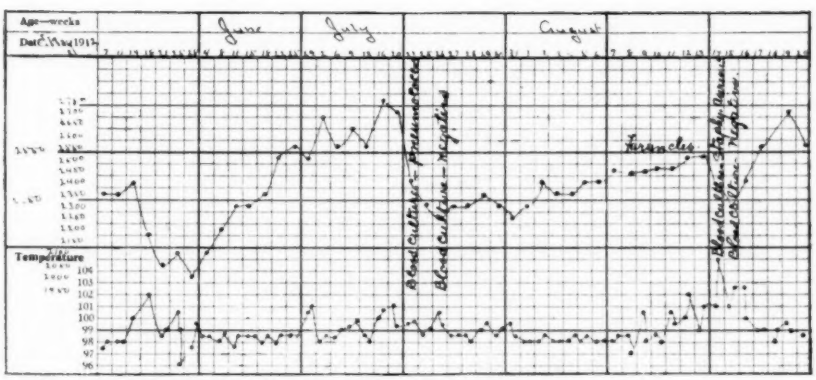
aureus. Both these invasions were unattended with definite clinical symptoms save the slight weight and temperature disturbance. During the last infection he had a few furuncles.

TYPES OF INFECTION

As previously mentioned in this paper, it is the pneumococcus and streptococcus infections that cause most of the trouble. Some idea of



Weight and Temperature Chart of Case 3.



Weight and Temperature Chart of Case 4.

the frequency with which these and other organisms are present in respiratory infections may be obtained from the results of a series of cultures made from the bronchial secretions of these infants. Altogether 619 sputum cultures have been made during the past three years in the

Babies' Hospital, and of these, 71 per cent. showed the presence in varying numbers of the pneumococcus, 42 per cent. the staphylococcus, and 31 per cent. the influenza bacillus. The staphylococcus was present in 85 per cent. of the cultures made and is the organism most frequently found in these infections. The variation in the organisms present may be seen in the following table, showing very little change from year to year:

	Pneumococcus Per Cent.	Streptococcus Per Cent.	B. Influenzae Per Cent.	Staphylococcus Per Cent.
A.—Jan. 31, 1911 to May 31, 1911	69	37	32	76
B.—Oct. 1, 1911, to May 31, 1912	82	46	33	89
C.—Oct. 1, 1912, to April 6, 1913	77	43	28	90

An examination of eighty-six cultures made from the pus after paracentesis of one or both ears of the infants gave the following results:

	Cases
B. Influenzae	6
Pneumococcus	15
Streptococcus	17
Staphylococcus	57

In a comparative study of cultures made from the sputum and nasopharynx some three years ago by Dr. Wollstein, at the Babies' Hospital, it was found that there was no essential difference in the variety of organisms in these different localities. If the pneumococcus was found in the nose and nasopharynx it was invariably present in the sputum culture. Apparently, these organisms are not limited to any special location in respiratory infections.

METHOD OF TRANSMISSION

It has long been believed that the common contagious diseases, scarlet-fever, diphtheria, whooping-cough, measles, small-pox, mumps and rubella are to a large extent communicated by aerial transmission. Of late, considerable experimental and clinical evidence gathered from abroad and in America has been brought forward to show that this is not the case. To Grancher of Paris is due the credit for first putting into practice prophylactic measures based on the assumption that infection was by contact. From his methods the plan has been developed of segregating the contagious diseases by means of separate stalls or cubicals, etc., and treating different types in the same ward with the assistance of aseptic nursing. Some disregard air separation; others aim to interrupt, partially or wholly, air exchange between compartments occupied by patients. All, however, seek to establish physical separation and to

prevent contact. The adequacy of these methods has been demonstrated both in Europe and in this country.

In respiratory infections, however, this plan does not offer anything like such a uniform success as has been obtained in the contagious diseases mentioned; if it were so, our problem would be much more simple.

Concerning the pneumococcus and streptococcus, the possibility of aerial transmissions seems greater, and to date there is lacking sufficient evidence to prove that such transmission does not occur.

During the past year there have been made in the Babies' Hospital some observations on ward atmosphere which bear on this point. Petri-plates with blood-agar media, were exposed in the wards when the air was quiet and during sweeping with sawdust dampened with 1 to 2,000 mercuric chlorid. At sweeping times the number of organisms in the air was greatly increased.

In every instance the plates were exposed five minutes. It is rather interesting to note that in the premature ward where the floor is wiped with damp cloths and where special care is taken to exclude every infant and every attendant suffering from any form of infection, there were usually found but one to two colonies. On one occasion, however, a number of colonies of pneumococcus were found. Two days later one of the premature infants developed a fatal pneumococcus meningitis.

PROPHYLAXIS

It is unnecessary to discuss measures of respiratory prophylaxis previously proposed, especially as regards the use of hexamethylenamin. None of these procedures have so far met with success. The use of hexamethylenamin as a respiratory antiseptic has been shown by Burnham to be valueless. The doses in which it can be given are not sufficient to exert any bactericidal action, and furthermore, the ability to be excreted as formaldehyd varies with the individual. Much that has been claimed for it was without justification.

Wollstein and Meltzer from their experiments on the production of pneumonia in animals by insufflation have reached the conclusion that the organisms enter from the upper respiratory passages and gradually find their way into the smaller bronchi where they multiply in this enclosed space and produce the lesions typical of pneumonia. They also showed that organisms injected into the blood do not produce pneumonia, but, on the other hand, they may be recovered from the blood in five hours after their introduction by insufflation. These observations are rather interesting from the clinical standpoint when one considers how frequently from a pneumonia patient one may obtain a history of a previous head cold. It has seemed to us that these observations suggest that preventive measures would give beneficial results in the treatment of all

ward cases. We will now pass to an account of our own attempt to prevent respiratory ward infections.

1. Increasing the child's individual resistance by careful attention to its nutrition.

2. By careful nursing for the purpose of minimizing the danger of transmitting infection.

3. By the direct prevention of the respiratory infections by postnasal douching.

The first of these measures has received our constant attention, while the second is almost an impossibility where nurse-maids do the greater part of the handling of patients. The third has been the object of our special study during the six months from January, 1913, to June, inclusive, and was undertaken from the experience of one of us (Chappell) in the treatment of colds, influenza and various infections of the upper respiratory tract for the past fifteen years. This treatment is postnasal douching with warm antiseptic solutions, chiefly boric acid, followed by argyrol solutions of various strengths. The cleansing of the nasopharynx of all mucus and food-remains, which form a good culture medium for the development of the various organisms, seems very desirable as a preventive measure in these cases.

Syringe for post nasal injections.

Two wards, A and B, were taken during the above period; forty patients were treated in each ward. In both these wards the floor was swept each morning with sawdust dampened with 1 to 2,000 mercuric chlorid solution, and each ward fumigated once a month. So far as could be carried out, no discrimination between types of babies admitted to the above-mentioned wards was made. Our only difference in treatment was that in Ward B, postnasal douching was carried out immediately on the patient's admission and subsequently twice a week.

The postnasal syringe used (Fig. 5) was devised by one of us (Chappell) and was of the same pattern as one used for older children and adults. It was, however, made much smaller. The syringe is made of metal having a capacity of 6 c.c. The openings at the tip of the syringe are arranged so that the fluid is directed upward, forward and backward, but not laterally, thus minimizing the risk of the entrance of fluid into the eustachian tubes. The advantages of the syringe are as follows: (1) The small quantity of solution used does not put any extra pressure in the naso-pharynx; (2) the small curve makes it very easy to

pass behind the curtain of the soft palate and cannot produce any traumatism, nor does it require great skill in its use; (3) the openings in the tip are on the anterior and posterior surfaces, and a transverse slit on the end with no lateral openings.

The infant is wrapped tightly in a sheet and held in a sitting posture on the lap of a nurse. While depressing the tongue the syringe is carefully introduced behind the soft palate in such a manner that the tip is held free in the postnasal space; the contents are then quickly discharged, the solution pouring out of both nostrils, mingled with curds and mucus. As soon as the contents of the syringe have been evacuated the nurse quickly throws the infant's head forward, in this manner facilitating the expulsion of mucus, etc. The crying of the infant has been found to be an advantage, since it aids in the more thorough cleansing of the nostrils. Occasionally, streaks of blood are obtained. This was more frequently seen when the infant had adenoids and was apparently due to the slight traumatism of the syringe, and did not occur when more experience was gained. In those patients in whom small amounts of blood accompanied the douching, no ill effects followed, either immediate or remote.

The chief objection urged against postnasal douching is the possibility of the introduction of fluid into the eustachian tubes. Our observations, however, do not indicate that, if the procedure is carefully carried out, the danger is great, for only four cases of otitis developed in the trial ward, while eleven developed in the corresponding ward where postnasal douching was not done. Although the number of cases is small, it should be considered that during this period over 400 postnasal douches were administered to infants; yet in private practice, on older patients many thousands of postnasal douches have been given by us without the development of any aural symptoms. It seems from this experience that the postnasal douching so far from being a cause of acute otitis, was in reality a valuable prophylactic measure. Throughout the period of observation the ears of all patients were carefully watched, even when no symptoms were present. A reduction in the number of pneumonias was also noted.

In totalling all the infections for the six months in Wards A and B, it was found that in Ward A there were fifty-seven infections and in Ward B (douching) only twenty-seven, a reduction of 50 per cent. The results can be more readily seen in the following table:

	Ward A	Ward B (Prophylactic douching).
Total patients treated	40	40
No. of infections present on admission	10	11
No. of infections developing in hospital	27	27

It is worth mention that in Ward B there developed two patients with retropharyngeal abscess and in Ward A there were none; whether the use of the postnasal syringe was a factor in these cases it is impossible to state.

During the six months of observation we used first a 1 per cent. boracic acid solution, followed immediately by a 1 per cent. argyrol solution. Of course, these solutions undoubtedly acted only as a wash; they certainly were not bactericidal, as cultures were made to ascertain this fact. More recently we have been employing undiluted hydrogen peroxid. The injection of this is followed by a solution composed of three quarters normal saline and one quarter Seiler's solution, with results that have appeared better than were obtained with the milder solutions. A larger experience is necessary to determine whether these stronger solutions are preferable to the milder ones. The fact that otitis has frequently been attributed to the use of peroxid has received our special attention. So far, ninety-six douches have been given in Ward B and no case of otitis has developed; while in Ward A, where none of the children received the douche, four cases have developed.

Cultures have been made before and after douching with peroxid and bacterial counts made from the washings of the postnasal space with sterile water. We have not been able to come to any conclusions on this point from the fact that the counts varied considerably and the reduction, when it did occur, was not sufficient to warrant definite conclusions.

The treatment by postnasal douching is now being continued by other observers at the Babies' Hospital and the results obtained so far confirm our reports.

PROGRESS IN PEDIATRICS

RECENT CONTRIBUTIONS TO OUR KNOWLEDGE OF NEPHRITIS *

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PRODUCTION OF NEPHRITIS

Dickson[1] has succeeded in producing a chronic diffuse nephritis in rabbits, guinea-pigs and dogs by the repeated administration of uranium nitrate. The histologic picture closely resembled that found in man except for the absence of demonstrable arterial lesions. In guinea-pigs death was usually due to an attack of acute nephritis, frequently accompanied by more or less anasarca. In the more severe cases there was usually found also some hypertrophy of the left ventricle of the heart.

O'Hare[2] has also produced a chronic nephritis by injecting living cultures of colon bacilli intravenously into animals under the influence of uranium nitrate. In all, he experimented on 40 rabbits, but excludes from further consideration 22 which died shortly after the first injection. The kidneys of 12 of the remaining 18 showed sufficient chronic change to justify the term chronic experimental nephritis. "The lesions so produced are closely similar to those involving the renal tissues in chronic interstitial nephritis in man." The kidneys of the remaining six rabbits showed no more change than is frequently found in the normal animal.

Christian and O'Hare[3] report on the glomerular lesions produced by acute uranium nephritis in the rabbit, and describe the following five forms: "There is a type of degenerative lesion evidenced mainly by the appearance of hyaline droplets in the capillary walls. Another type of lesion consists in the formation of fibrin thrombi in the capillaries. A third type is hemorrhagic in nature and appears usually as a rounded mass of fibrin enmeshing blood-corpuscles. A fourth type is a dilatation of the glomerular space with granular material. A fifth type is a proliferative lesion affecting chiefly the capillary endothelium, and to a much less extent the capsular epithelium."

* Submitted for publication March 1, 1914.

1. Dickson, E. C.: Arch. Int. Med., 1912, ix, 557.
2. O'Hare, J. P.: Arch. Int. Med., 1913, xii, 49.
3. Christian, H. A., and O'Hare, J. P.: Jour. Med. Research, 1913, xxiii, 227.

O'Hare[4] has compared the lesions produced by uranium nitrate on dogs and on rabbits, and concludes that the dog is much more resistant to the poison, as a dose of uranium nitrate three and a half times as large per kilo of body weight as the one commonly employed in the rabbit produced no more extensive lesions. "The glomerular lesion with the formation of hyaline droplets is produced with great difficulty in the dog."

Baehr[5] injected uranium nitrate in 0.35 to 0.8 mg. doses directly into the renal arteries of rabbits and found far more severe changes in the glomeruli than he did in the tubules. From 40 per cent. to 50 per cent. of the glomeruli were affected. Subcutaneous injections produced a similar but less marked result. Injections of 1 c.c. of a 2 per cent. solution of potassium chromate caused a high grade necrosis of the tubules, especially of the first and second parts of the tubuli contorti. One drop of croton oil diluted with fifty drops of olive oil, emulsified in 1 c.c. of distilled water and injected into the renal artery, led to severe glomerular changes with the formation of hemorrhagic cysts. A 0.02 to 0.05 per cent. aqueous solution of iodin produced a contracted kidney with secondary vascular sclerosis, without affecting the glomeruli.

MacNider[6] made a careful anatomical study of the effect of the various kidney poisons on dogs, and decided that cantharidin, potassium dichromate, uranium nitrate and sodium arsenate all produce an acute nephritis by affecting first the vascular elements of the kidney and then the tubular. Uranium nitrate and potassium dichromate usually caused tubular changes much earlier than did cantharidin or sodium arsenate. In the early stages of the nephritis when the kidney under the microscope showed only vascular changes, the output of urine was increased. Then as the tubules became involved the amount of urine diminished. All the animals which had anuria showed also varying degrees of obstruction of the lumina of the tubules by desquamating epithelium. None of this was found in the animals who were killed during the stage of polyuria. Pearce[7] considers that cantharidin and arsenic cause a vascular nephritis characterized by marked changes in the power of the kidney to respond to such stimuli as adrenalin or diuretin; and that potassium chromate, uranium nitrate and corrosive sublimate belong to another type with striking histological changes, namely, a tubular nephritis. Nevertheless, neither type can be obtained pure; for instance, in cantharidin nephritis, after the third day, many mitotic figures can be found in the tubular epithelium. Hence "great caution must be observed in ascribing

4. O'Hare, J. P.: Arch. Int. Med., 1913, xii, 61.
5. Baehr, G.: Beitr. z. path. Anat. u. z. allg. Path., 1913, lvi, 544.
6. MacNider, W. deB.: Jour. Med. Research, 1912, xxi, 79.
7. Pearce, R. M.: Jour. Exper. Med., 1913, xvii, 542.

the physiological disturbances of kidney function caused by cantharidin as due to a vascular injury and in regarding cantharidin nephritis as a pure type of vascular nephritis."

The action of tartrates on the kidney has also been studied by Pearce.[8] He has given the salt to dogs by mouth, intraperitoneally and subcutaneously, and finds that it produces necrosis of the convoluted tubules with fatty changes in the loops of Henle, and sometimes also in the collecting tubules. Exudative glomerular lesions occurred in about half of the severe cases. The mode of administration was without influence on the result. The urine is much diminished, is clear, of low gravity; and contains albumin and casts. Underhill, Wells and Goldschmidt[9] found that sodium tartrate injected subcutaneously into rabbits failed to appear in the urine and concluded that the disintegrative effect of the salt on the convoluted tubules was sufficient to prevent its elimination. Evidently the glomeruli were unable to take over this function of the tubules. The same authors[10] have also found that the administration of an alkali by mouth (sodium carbonate or citrate) during tartrate nephritis slightly favors the elimination of the urinary constituents and also has a slightly beneficial effect on the pathological changes in the kidneys.

Pearce[11] has found that crotalus venom caused an acute exudative glomerular nephritis in rabbits characterized by a persistent albuminuria and cylindruria, emaciation and death in five or six weeks. He was unable to produce any subacute or chronic glomerular lesions by this means.

Hirsch and Maschke[12] in the course of experiments on dogs, the details of which are not given, found that intraperitoneal injections of egg-white did not cause severe kidney degeneration. They state that by staining histological preparations of the kidneys they were able to trace the excretion of the egg white and that it was excreted through the glomeruli and not through the tubules.

Longcope[13] has studied the action of foreign proteins on the kidney. He worked with horse serum and egg white on a large series of dogs, cats and rabbits, giving first a small sensitizing injection, and later repeated small doses insufficient to cause symptoms of anaphylactic shock. He found that changes were especially marked after peritoneal injections in the peritoneum and after intravenous injections in the liver of rabbits

8. Pearce, R. M., and Ringler, A. I.: Jour. Med. Research, 1913, xxiv, 57.
9. Underhill, F. P., Wells, H. G., and Goldschmidt, S.: Jour. Exper. Med., 1913, xviii, 317.
10. Underhill, F. P., Wells, H. G., and Goldschmidt, S.: Jour. Exper. Med., 1913, xviii, 322.
11. Pearce, R. M.: Jour. Exper. Med., 1913, xviii, 149.
12. Hirsch, C., and Maschke, W.: Berlin. klin. Wchnschr., 1912, xlix, 145.
13. Longcope, W. T.: Jour. Exper. Med., 1913, xviii, 678.

and cats, and in the myocardium and kidneys of all groups of animals. In dogs and rabbits there developed a well-marked nephritis characterized by degeneration and necrosis of the epithelium of the loops of Henle, of the collecting tubules, and less frequently of the convoluted tubules. There were also acute and chronic alterations in the glomeruli of all groups of animals. He decides that "though it is impossible at the present time to say definitely that the development of nephritis depends exclusively on the previous sensitization of the animal to these substances (the foreign proteins), it seems probable that at least any primary toxicity of the protein for the kidney cells was heightened by this procedure, and that the lesions in the kidney as well as those in the liver, myocardium and peritoneum are to be interpreted as a generalized reaction analogous to the local 'Arthus phenomenon' in the skin of the rabbit." He makes no mention of the production of fever as a result of the repeated protein injections.

PROTEIN AND THE KIDNEY

The retention of foreign proteins by the kidney has been studied at length by Pearce.[14] He injected a series of rabbits with egg white or horse serum, killed them at varying intervals after the injection and made extracts of their kidneys. He concludes that "extracts of the kidneys of normal rabbits prepared one, two, three and four days after the intravenous injection of egg-white and horse serum have the power to sensitize guinea-pigs to a second injection of these proteins. The sensitization by first- and second-day extracts was constant and intense, that by the third-day extracts was less marked and sometimes was not evident, and that by the fourth-day extracts was only occasional, and when present was always weak. Comparative studies of the power of the blood, liver and kidney to sensitize indicate that this sensitization depends on the content of the foreign protein in the circulating blood and not on its accumulation or fixation in the tissues of an organ. This opinion is supported by other experiments in which the sensitizing power of the blood and of the extracts of unmarked kidneys was compared with the sensitizing power of marked kidneys. The weak sensitizing power of washed kidney extracts is taken as evidence that foreign proteins of the kind used are not held in the tissues of the kidney, and, if these results may be applied to nephrotoxic proteins, it follows that nephritis is not due to selection and persisting fixation of a protein by the renal cells, but is due to the action of such proteins merely during the process of elimination. In experimental acute nephritis of the type due to uranium nitrate, the power of sensitization to egg albumin is prolonged for

14. Pearce, R. M.: Jour. Exper. Med., 1912, xvi, 349.

twenty-four hours, and in the chromate type for forty-eight hours, thus indicating that in nephritis of the acute type at least the elimination of a foreign protein is delayed." His attempts to obtain sensitization in this way with vegetable or bacterial proteins failed.

Vaughan[15] comments on this as follows: "In our opinion the possibility of harm coming to the kidney or any other organ from the deposition of a foreign protein in it is not due to any directly poisonous effect of the foreign protein, but to the liberation of the poisonous group when the body cells become sensitized and split up the foreign protein."

Folin[16] has applied his new methods for nitrogen determination in the blood to cats suffering from various forms of acute experimental nephritis. In the tubular type, as illustrated by chromate nephritis, there was only moderate nitrogen retention; whereas in uranium and cantharidin nephritis, in which both tubules and glomeruli are involved, the additional lesions in the glomeruli led to a marked retention of nitrogenous waste products.

CHLORID IN NEPHRITIS

Borchardt[17] concludes from a review of the literature that sodium chlorid is excreted through the glomeruli by filtration and then concentrated by the reabsorption of the water during its passage through the tubules. When the tubular epithelium is injured the salt also is reabsorbed, so that the urine cannot be concentrated. In the acute stage of glomerular disease there is chlorid retention because of the lessened area of filtration. On the other hand, in a mild acute glomerulonephritis, or during the chronic stage, the chlorid excretion is normal. In general, "a normal sodium chlorid excretion speaks strongly against a severe disease of the kidney epithelium."

Ambard and Weill[18] consider that the concentrations of urea and chlorid in the urine, and the total amounts of each excreted, bear a certain definite relation to each other, both in health and in disease. If this is true, and if it is granted that some nephritis is primarily tubular and some primarily glomerular, then the prevailing view, that chlorids are excreted by the glomeruli and urea by the tubules, must necessarily be false. Their argument is based on clinical studies of nephritis in man.

Widal, Amberg and Weill[19] have studied the influence of a salt-free diet on the threshold for the excretion of chlorids by the kidney. They

15. Vaughn, V. C.: Protein Split Products, Lea & Febiger, Philadelphia, 1913, p. 363.
16. Folin, O., Karsner, H. T., and Denis, W.: Jour. Exper. Med., 1912, xvi, 789.
17. Borchardt: Deutsche Med. Wchnschr., 1912, xxxviii, 1723.
18. Ambard, L., and Weill, A.: Semaine méd., 1912, xxxii, 217.
19. Widal, F., Amberg, L., and Weill, A., Semaine Med., 1912, xxxii, 361.

found that in cases of nephritis with edema the threshold was considerably raised and that there was a more or less marked retention of chlorids. By giving or withholding salt in the diet they were able in most cases to raise or lower the threshold at will and simultaneously to cause the appearance or disappearance of the edema. In cases of nephritis without edema they found no retention of the chlorids.

DIURETICS IN NEPHRITIS

Boycott and Ryffel[20] have compared the effects of the so-called "secretory" and "mechanical" diuretics in acute experimental nephritis in rabbits. The former are supposed to act on the tubules and the latter on the glomeruli. As representative of the first class the authors used caffein and of the second class Ringers solution and 5 per cent. sodium chlorid solution. They produced a tubular nephritis by the subcutaneous injection of uranium nitrate. Theoretically, caffein should then "fail to excite the secretion of urine while Ringer's fluid or 5 per cent. salt solution should still be effective diuretics." Contrary to their expectation, the authors found that "in any case where a 'secretory' diuretic has failed, 'mechanical' diuretics have also failed, and where the one sort has been effective the other sort has acted equally. The response of the normal kidney to caffein is slower than to the other diuretics used. The response of the nephritic kidney to all three diuretics is slower than that of the normal kidney, and the response to caffein remains slower than to Ringer's or salt solution. The urine produced by the kidney in the early stages of uranium nephritis is smaller in amount and contains less chlorid than normal. Caffein urine in normal animals contains less chlorid than that produced by the injection of Ringer's or salt solution; in nephritic animals all three urines contain less chlorid than normal, and caffein urine still contains less than the other two. In the course of the response to any one diuretic the concentration of chlorid in the normal urine increases as the rate of secretion decreases; in the nephritic urine there may be a very slight increase in concentration, or the concentration may fall as the rate of secretion becomes less. Uranium causes glycosuria; the method of experiment also causes glycosuria in normal rabbits. This has to be taken into account in considering the variations in the concentration of chlorid."

These experiments were performed on rabbits. Previous to this, MacNider[21] had done somewhat similar work with dogs. He found that early in a uranium nephritis they developed polyuria and glycosuria.

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20. Boycott, A. E., and Ryffel, J. H.: Jour. Path. and Bact., Cambridge, 1913, xvii, 458.

21. MacNider, W. deB.: Jour. Pharm. and Exper. Therap., 1912, iii, 423.

The administration of an anesthetic at this stage rendered them partly or completely anuric. "Such animals, under the effect of caffein, theobromin, digitalin and 0.9 per cent. salt solution, show a normal response in the blood-pressure rise and in the vascular response of the kidney (measured by oncometer)." Some of the animals responded to the diuretics used, while others, under exactly similar conditions, remained anuric. There was no difference in the vascular response of the kidneys of the two groups. Histologically, however, the tubules of the animals which remained anuric were found to show much more severe epithelial lesions and to be practically blocked. MacNider considers that "this investigation indicates that epithelial changes are more responsible for the production of anuria than are the vascular changes."

Christian[22] reviews the work done at his laboratory on the action of diuretic drugs in acute experimental nephritis. He and O'Hare[23] produced a severe uranium nephritis in eighty-two rabbits, half of which were then given theobromin, while the rest were kept as controls. "The nephritis . . . was fatal in all except twelve rabbits, nine of which had received theobromin sodium salicylate. Of those receiving theobromin sodium salicylate by stomach tube and dying, the average duration of life was 6 days; of controls 6.94 days. Of those receiving theobromin sodium salicylate intravenously the average duration of life was 4.56 days; of controls 6.23 days. Thus under the conditions of our experiment theobromin sodium salicylate materially shortened the life of animals with severe acute fatal nephritis." Walker and Dawson[24] under Christian's direction, performed similar experiments on rabbits with other drugs. "The average duration of life was as follows: caffein, 5 days; theocin, 4 4/7 days; potassium acetate, 4 4/7 days; spartein sulphate, 6 days, with one surviving animal, while control rabbits lived 8 1/5 days. These results are essentially similar to those obtained with theobromin sodium salicylate." Water alone, given intravenously, "produced but little change in the average duration of life." Christian[22] also reports on the phenolsulphonephthalein excretion of rabbits with acute uranium nephritis. "The excretion of phenolsulphonephthalein was measured before and after giving the uranium. There was a prompt decrease in the ability of the kidney to excrete this coloring matter after uranium had been given. This decrease in phenolsulphonephthalein excretion in rabbits having a very severe nephritis appeared to be uninfluenced by theobromin sodium salicylate." In a somewhat milder nephritis "the phthalein output was less markedly decreased in the animals receiving theobromin sodium salicylate than in those not receiving the drug."

22. Christian, H. A.: Jour. Am. Med. Assn., 1913, lxi, 267.
23. Christian, H. A., and O'Hare, J. P.: Arch. Int. Med., 1913, xi, 517.
24. Walker, C., and Dawson, R. P.: Arch. Int. Med., 1913, xii, 171.

Sellei[25] has reported on the action of theocin in human beings. He used the sodium acetate in 0.3 gm. doses. With normal kidneys there was a diuresis lasting three to four hours. The specific gravity and chlorids of the urine were unchanged and the phosphates were slightly diminished. In pyelitis the result was the same as in the normal kidney. In nephritis there was usually diuresis, the specific gravity was lowered, the chlorids were diminished but the phosphates were usually increased.

Picot[26] describes three cases of oliguria in which he produced diuresis by giving 25 gm. of urea in a glass of milk.

Widal, Amberg and Weill,[19] in connection with their work on the threshold for the excretion of chlorids, report that theobromin has the same effect as a salt-free diet and produces a marked reduction of the threshold. Its action is directly antagonistic to that of sodium chlorid.

KIDNEY FUNCTION

There have been many attempts to correlate the various tests of renal function with the different forms of nephritis, both clinical and experimental, but so far none have been very successful. The difficulty lies partly in producing experimentally a purely tubular or purely glomerular nephritis, and partly in the fact that the great majority of cases of nephritis in man are of a mixed type. Fitz and Rowntree[27] state that they "have considered the excretion of lactose as an index of vascular functional capacity; that of phthalein as an index of total renal function (though predominantly tubular); and the excretion of a salt and iodid as an index of tubular functional capacity." They produced kidney lesions in rabbits by clamping off the renal circulation for varying periods of time and found that "except in the most extreme cases, there is no definite relation possible by the functional tests used between the pathological and the functional disturbances produced.

Baright[28] makes use of the accompanying table and attempts to fit his patients into one of the three groups, but is forced to admit that most of them are mixed types. He states that nephritis with delayed lactose excretion is common, comprising 40 to 50 per cent. of the cases. Cases of urea retention and of delayed iodid and chlorid excretion are rare in pure form. "Acute beginning nephritis is usually a mixture of the three types, with lactose retention, strong potassium iodid and urea retention, some increase in blood-pressure and perhaps hematuria — glomerular irritation; oliguria and edema — tubular injury, and uremic troubles due to accumulation of nitrogenous wastes in the blood — not necessarily

25. Sellei, J.: Ztschr. f. Urol., 1912, vi, 264.
26. Picot, G.: Jour. d'urol. méd. et chir., 1912, ii, 203.
27. Fitz, R., and Rowntree, L. G.: Arch. Int. Med., 1913, xii, 24.
28. Baright, H. E.: Med. Rec., New York, 1913, lxxxiii, 699.

urea. It may heal or may resolve into chronic vascular, chronic tubular or chronic uremic type."

TABLE OF DIFFERENT FORMS OF NEPHRITIS AS DETERMINED BY FUNCTIONAL TESTS

Experimental Type / Clinical Type		Cantharides Vascular	Corrosive Tubular	None Uremic
Function Tests				
Excretion Time	Normal			
2.0 gm. lactose	.4°	5-12°	4°	4°
0.5 gm. potass. iodid	40°	40°	80-100°	40°
10.0 gm. sod. chlorid,	24°	24°	72°	24°
10.0 gm. urea	24°	24°	24°	Retained

Conzen[29] has tried the lactose and potassium iodid tests on 37 patients who later came to autopsy. He states that the tests were a great aid in diagnosis. "The intake of 10 gm. salt increases the albuminuria and leads to water retention and general malaise only in true nephritis and in severe congestion of the kidney."

Normenbruch,[30] from a study of clinical cases, concludes that the congested kidney functionally resembles a pure nephritis, approaching the tubular variety in chlorid excretion and the severe vascular variety in water excretion. Nitrogen excretion is fairly good, thus differing from the cases of true vascular nephritis. Nevertheless there is some nitrogen retention whenever the oliguria becomes marked.

Wolfheim[31] considers that it is possible to differentiate the different forms of nephritis by means of functional examinations, basing his opinion on the study of 28 cases on a strict milk or milk and rice diet, with or without the addition of given amounts of salt or of water. He found normal nitrogen elimination in acute nephritis. There was salt retention in the oliguric stage of both vascular and tubular nephritis. Edema did not necessarily follow retention of chlorid, but all cases of edema showed some chlorid disturbance either from retention or from the flushing out of retained deposits.

Frothingham, Fitz, Folin and Denis[32] have made a comparative study of the relation between the non-protein nitrogen retention and the phthalein excretion in uranium nephritis using the new methods for the determination of the nitrogen in the blood. They reached the following conclusions: "In acute uranium nephritis in rabbits the excretion of phthalein in the urine and the amount of non-protein nitrogen and urea in the blood vary from the normal during the course of the nephritis and return to normal as the nephritis heals. The degree of variation from

29. Conzen, F.: Deutsch. Arch. f. klin. Med., 1912, cviii, 353.
30. Normenbruch, W.: Deutsch. Arch. f. klin. Med., 1913, cx, 162.
31. Wolfheim, W.: Ztschr. f. klin. Med., 1913, lxxvii, 258.
32. Frothingham, C., Fitz, R., Folin, O., and Denis, W.: Arch. Int. Med., 1913, xii, 245.

the normal agrees on the whole with the amount of destruction demonstrated histologically in the kidney. The phthalein excretion in the urine drops rapidly to its lowest point and returns rapidly to normal with the recovery of the kidney. Non-protein nitrogen and urea accumulate gradually in the blood and return to normal gradually as the kidney recovers. In general, the tests parallel each other as indicators of renal function, but have this essential difference: the amount of phthalein excretion shows the renal function at the moment; the amount of non-protein nitrogen and urea in the blood is rather a measure of an accumulating difference between the amounts of waste nitrogen produced in the metabolism and the amounts eliminated by the kidneys. The time elements, the duration of the condition, is therefore an important factor in this test."

ACTION OF "OTHER THINGS" IN NEPHRITIS

MacNider[33] gave uranium nitrate to dogs in doses of 6 to 7 mg. per kilo of body weight and produced an acute nephritis in which both the vascular and the epithelial element of the kidney is involved, the latter rather more than the former. He found that the older the animal the more severe were the changes. "Following the use of an anesthetic these changes are increased in severity. The degenerative changes following the use of an anesthetic are also more pronounced in adult animals than in puppies. Of the anesthetics which have been employed in this investigation, Grehant's anesthetic, chloroform and alcohol, is more toxic than is morphin-ether."

Previous to this, Apperly[34] had studied the effect of chloroform on the kidney and decided that it did as much damage to the tubular epithelium as it did to the liver where its toxic action had long been known. He states that ether produces not nearly the same effect.

Underhill[35] has confirmed the observation of Baer and Blum that sodium tartrate given subcutaneously greatly diminishes the output of nitrogen and dextrose in the urine of phlorizinized dogs but considers that it is due to the acute tubular changes with anuria brought on by tartrate. He states that the phlorizin contributes nothing to the nephritis.

Hess and Wiesel[36] found lesions in the adrenals of dogs dying of uranium nephritis and hence were led to try the effect of the administration of adrenalin during the course of the nephritis. They conclude that it is possible to preserve life in uranium-poisoned dogs by simultaneous injection of epinephrin, provided that the latter is given before the

33. MacNider, W. deB.: Jour. Med. Research, 1913, xxiii, 403.
34. Apperly, R. E.: Brit. Med. Jour., 1912, ii, 624.
35. Underhill, F. P.: Jour. Biol. Chem., 1912, xii, 115.
36. Hess, L., and Wisel, J.: Wien. klin. Wchnschr., 1913, xxvi, 317.

onset of anuria, even if an almost invariably fatal dose of uranium has been given. At the same time they observed a decrease of albumen and an increase in the amount of urine — signs of a functional improvement. Anatomically, however, the renal lesions resulting from uranium cannot be influenced by epinephrin.

Georgopulos[37] produced uranium nephritis in eighteen dogs, half of which had been thyroidectomized, and found that the thyroidectomized animals lived from one to three days longer than the controls. He believes that this was due to hyperfunctioning of the parathyroids following thyroidectomy and suggests that the parathyroid secretion counteracts the systemic intoxication in nephritis and thus an extract of these glands might prove useful in the treatment of nephritis in man.

MISCELLANEOUS

Baehr[38] has reported the results of histological study of the renal lesions in human cases of endocarditis due to various organisms. He reached the following conclusions: "In most cases of subacute bacterial endocarditis due to the endocarditis coccus, there exists a distinctive glomerular lesion due to bacterial emboli. There is an involvement of one or more loops of a variable number of glomeruli, absence of disease in other glomeruli, and association of all stages in one microscopic section. The lesion does not occur in cases of acute endocarditis, or in subacute cases due to other bacteria. Healed cases of subacute endocarditis show the healed stage of the distinctive glomerular lesion, although it is less extensive than in the active bacterial cases. These cases are probably those in which the endocardial vegetations have become bacteria-free early in their course and are now healing or healed. During the active stage of the disease, if the emboli are not too numerous, the only sign will be an almost constant slight hematuria. More marked cases may show a hemorrhagic nephritis. Numerous glomerular lesions with recovery may lead secondarily to a contracted kidney."

Underhill, Wells and Goldschmidt[39] gave a mixture of sodium chlorid and urea intravenously to rabbits during tartrate nephritis and found that the extra salt reappeared in the urine within forty-eight hours, while there was no increase in the urinary nitrogen. In view of the primarily tubular character of tartrate nephritis this result may be taken as strongly confirmatory evidence of the old theory that chlorids are excreted by the glomeruli and urea by the tubules.

37. Georgopulos, M.: Ztschr. f. klin. Med., 1912, lxxvi, 261.
38. Baehr, G.: Jour. Exper. Med., 1912, xv, 330.
39. Underhill, F. P., Wells, H. G., and Goldschmidt, S.: Jour. Exper. Med., 1913, xviii, 347.

Kraus[40] has carried out very thorough metabolism experiments on four dogs with uranium nephritis. He found that in the normal animal any creatinin injected was excreted "in toto" and did not affect the other urinary constituents. Creatinin injected during acute uranium nephritis caused both the endogenous and the injected creatinin, and uric acid, chlorids and water, to be excreted in decreased amounts, and sometimes caused death. Inasmuch as fever increases the amount of endogenous creatinin, the onset of fever in a case of acute nephritis may well overtax the kidney and cause death.

The relation between the urea and the total nitrogen in the urine, known as the nitrogen coefficient, has been used as a diagnostic aid. Boulud[41] has carried out some experiments on dogs with corrosive, chloroform and quinin nephritis, and found a nitrogen coefficient as low as 60. Normally, in both men and dogs, it is 80 to 82. Furthermore, in cases of human nephritis he found a similar decrease, and in the cases of albuminuria where the nitrogen coefficient was normal, some other cause for the albumen always could be found.

Opie[42] tries to explain the occurrence of edema in nephritis as the result of changes in the blood-vessel walls produced by the same substance which causes the nephritis. In support of his opinion he brings forward the following experimental observations. Cantharidin in addition to causing nephritis, increases the flow of lymph and simultaneously blocks the lymph channels of the liver by injuring the lining cells and producing a fibrinous coagulum. Hence there develops well-marked edema of the liver coincident with and not as a result of the nephritis. Opie, however, makes no mention of the occurrence of edema during cantharidin poisoning elsewhere than in the liver.

The subject of the formation of renal casts has been taken up by Erdman.[43] As the result of microscopic and clinical studies, he believes that the hyalin casts are moulded from mucus in the tubules. The amount of mucus is increased in a concentrated acid urine and diminished in a dilute alkaline urine. Cylindroids he considers to be hyalin casts in the making. Granular casts may be formed in any one of three ways; either from the degeneration of the epithelial cells of the convoluted tubules, or from exudative material from the capsular spaces, or from the granular disintegration of coagulated blood.

The amount of permanent damage done by an attack of acute nephritis in childhood has been investigated by Ernberg.[44] He succeeded in

40. Kraus: Arch. Int. Med., 1913, xi, 613.
41. Boulud, R.: Lyon méd., 1912, cxix, 505.
42. Opie, E. L.: Jour. Exper. Med., 1912, xvi, 831.
43. Erdman, H. B.: Jour. Am. Med. Assn., 1912, lix, 1952.
44. Ernberg, H.: Nord. med. ark., 1911, xliv, 109.

locating and examining 40 out of 106 adults who had had acute nephritis before the age of 15 years, mostly in connection with an acute infection, and found normal urines in all. Furthermore, he examined 16 out of 50 adults whose acute nephritis had occurred between the ages of 15 and 30 years, and found that all were normal except 4. Two of these gave evidence of nephritis of recent origin; the other 2, whose original attack had been subacute rather than acute, showed albuminuria. If these figures give us a true picture we certainly have been unduly pessimistic about the prognosis of acute nephritis in childhood.

Pollitzer[45] considers that a slight opalescence or turbidity in urine following the addition of acetic acid indicates the presence of chondroitin sulphuric acid. He has found it present in cases of tonsillitis, arthritis, chorea and endocarditis preceding the development of albuminuria and lasting after the albumin had disappeared. He has also found it constantly present in orthostatic albuminuria and believes it to be a sign of damage to the kidney.

Quinan[46] has made quantitative analyses of the lipoids of the blood-serum and reports that in chronic parenchymatous nephritis they are increased up to 2.6 per cent. from a normal of about 1 per cent.

Fischer's colloidal theory of nephritis has been generally discredited as being based on insufficient evidence. The recent literature on the subject has brought forward no new arguments.[47]

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45. Pollitzer, H.: Deutsch. med. Wchnschr., 1912, xxxviii, 1538.
46. Quinan, C.: Cal. State Jour. Med., 1912, x, 64.
47. Moore, A. R.: Jour. Am. Med. Assn., 1912, lix, 423; 1913, lx, 345.

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